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APHASIA AND THE CEREBRAL ZONE OF SPEECH.[1]

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The cerebral zone of speech includes an extensive cortical region, probably not less than one-third of the cortical expanse of the lateral aspect of the left hemicerebrum, as shown in the shaded portion of Fig. 1. While for descriptive purposes it is best to designate it as a cortical area, it must be understood that both the cortex and subcortex of the entire region enter into its formation.

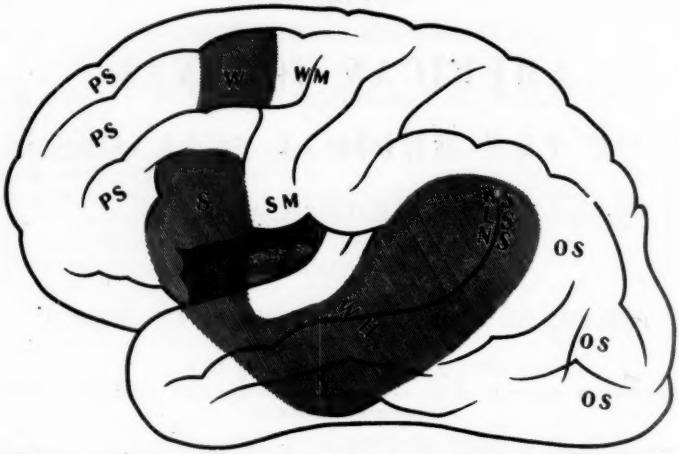
Within this great zone are several important centres or subareas concerned with particular attributes of language. In Fig. 1 these centres are *A*, a centre for word-hearing, the auditory portion of the zone of language; at *B* and *b*, a great centre for speech, often called the motor centres; at *V*, a visual subarea concerned with the representation of words, letters, and numbers; at *G*, a graphic or writing centre, usually spoken of as the motor graphic centre; and at *N*, a naming centre in which it is believed by those who hold to its existence that names are correlated with the concrete concepts of the objects named. In the diagram Fig. 2, in which the most important of the fundamental physiological areas of the cerebrum are represented graphically, these centres in the zone of language are more definitely isolated and designated by the terms word-hearing, speech, word-seeing, letter-seeing, number-seeing, writing, and naming.

Briefly stated, the auditive centre for speech is situated at the junction of the first and second temporal gyres about opposite the point where the horizontal branch of the Sylvian fissure turns upward posteriorly; the dynamic or motor speech centre occupies

[1] A paper presented to the College of Physicians of Philadelphia, June 1, 1904.

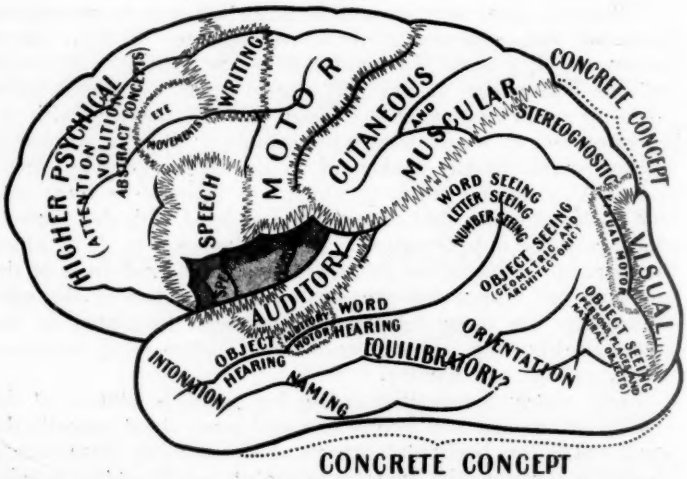
the hinder portion of the subfrontal or third frontal convolution, and, according to the views of the writer, the insula or at least its

FIG. 1.



Cerebral zone of language: *W*, writing centre; *S*, *S*, speech centre (language arrangement for emission); *WH*, word-hearing or auditory centre; *WS*, word-seeing; *LS*, letter-seeing; *NS*, number-seeing; *N*, naming centre; *PS*, *PS*, *PS*, higher psychic region; *WM*, centre for movements of upper extremity concerned in writing; *SM*, centre for movements concerned in speaking; *OS*, *OS*, *OS*, area for object-seeing.

FIG. 2.



Diagrammatic scheme of the physiological areas and centres of the lateral aspect of the human hemicerebrum.

anterior half; the visual centres immediately concerned with speech are located in the angular gyre; the site of the graphic or writing centre is in the caudal half or third of the second frontal convolution, and the naming centre is probably situated in the third temporal convolution.

Connecting these centres with each other, and also with other portions of the brain, are subcortical tracts composed of the processes of the cell bodies which are located in the cortex and of their fibrillary constituents. These associating tracts are of a complicated kind, although their number and arrangement are capable of analysis if sufficient time and study are given to the matter. Only one or two points regarding them need here be emphasized, namely, that the different centres are connected each to the other by a system of to-and-fro association, so that the impulses from special areas probably pass in both directions.

Surrounding the zone of language on all sides are centres and areas which have important relations to the function of speech. These are of at least three kinds, and an understanding of them may be reached by a study both of Fig. 1 and Fig. 2. On the sensory or receptive side of the brain are areas or centres concerned with such functions as object-seeing and object-hearing. Objects which are not recognized cannot of course be named, and these centres for object-recognition, therefore, are necessarily contributors to the sensori concept side of the zone of language. They are represented in Fig. 1 at the points marked as *OS*, and in Fig. 2 by the designation object-seeing, with subareas for geometric and architectonic forms, and for persons, places and natural objects. On the motor or emissive side of the cerebral speech zone are centres at *SM* and *WM* which are concerned with the execution of the work prepared for them respectively by the speech and writing centres. They are, in other words, the centres of representation of movements of articulation and enunciation, and of the movements of the hand and upper extremity generally. At *PS* in the prefrontal portion of the brain is a region where the highest psychical functions are especially represented. It is evident that in using language, attention, volition, and reasoning play a part, although this part may vary within wide limits. This higher psychical region, therefore, has its particular relation to the zone of language and especially to its dynamic side.

Broad, and it may be somewhat loose, comparisons may be helpful to the understanding of an intricate subject. The zone of language and that from which it is derived and with which it is correlated may be compared to the machinery and the methods by which one of our great natural products, like oil, is handled. The centres for object-recognition and the primary percept centres through which their memorial images are obtained may be compared to the producers or feeders of the system, the zone of language

with all its centres and their connections to the refiners of the raw product, the executive motor centres and their projection fibres to the distributers, while the higher psychical region is the suzerain or director of the entire business.

My objects in this paper are (1) to present succinctly a series of personal observations, some already recorded, and others not, demonstrating the existence of the centres of the zone of language according to the exposition just made, in this connection giving some corroborative proofs, although any extensive study of the enormous literature of the subject will be avoided; (2) to show by personal clinicopathological cases, with necropsies, the correlations of the zone of language with the sensory and motor spheres of the brain; and (3) leaving clinicopathological facts, to give briefly the biological principle which is fundamental to our conception of the speech zone and its subdivisions. That a discussion such as is intended in this contribution is called for at this time is evidenced in the first place because the extent and the particular subdivisions of the zone of language are still matters of dispute among those who have paid particular attention to cerebral speech. As high an authority in neuroanatomy and in clinical neurology as Dejerine, for example, still contends that a graphic centre in the second frontal gyre does not exist, holding that Broca's convolution is sufficient for all the purposes of the arrangement of language both for articulatory expression and for writing. The question of a naming centre may be regarded as still under discussion. Another matter of some interest and of some uncertainty is the exact part played by the subfrontal gyre and the insula respectively in the mechanism of language, whether, for instance, these parts represent stages in the evolution of cerebral language or whether they act conjointly as one centre. The writer has had the unusual opportunity of observing clinical cases with necropsies and in some instances cases operated on by surgeons, which demonstrate every localization given in the zone of speech as here delineated and discussed. In presenting these cases I shall condense as much as possible, being desirous only of enforcing particular views without going into cumbersome details. References made to cases hitherto recorded will be especially brief. It will appear that however much I may have used the work of others in arriving at conclusions regarding the part played by the cerebrum in the faculty of speech, I have in every case been able to confirm the positions adopted or assumed by personal experience. In taking up the discussion of the different centres I shall proceed in part chronologically and in part in obedience to the natural development of the subject, first directing attention to the speech centre which has been longest known and is in most respects at least of greatest importance, that which is, in the writer's opinion, located conjointly in the subfrontal convolution and in the insula.

Two photographs (Figs. 3 and 4) are given of a case which has scarcely been exceeded in value in corroborating the existence of a

FIG. 3.

Lateral aspect of the left hemicerebrum, showing one portion of an old lesion circumscribed to the hinder part of the third convolution.

FIG. 4.

Horizontal transection of the brain, showing lesion of the insula continuous with that in the third frontal convolution indicated in Fig. 3; also atrophy of the entire left hemisphere, with enlargement of the ventricles.

centre or area of representation of speech in the caudal portion of the third frontal gyre and in the insula. Gordinier has referred to this case in one of his papers on agraphia, but without illustration, a photograph of the exterior of the left hemisphere and a brief history of the case having been furnished him by the writer.[1] At *A* in Fig. 3 is shown the external portion of an old hemorrhagic cyst, the only lesion related to the aphasia of the patient to be seen on the lateral aspect of the hemicerebrum. It will be observed that this area is strictly confined to the caudal portion of the subfrontal convolution. This brain has been sectioned only recently. A horizontal cut was made at the level of the upper surface of the insula, and revealed the lesion and the appearances shown in Fig. 4. It will be seen that the insula is largely destroyed. This proved on investigation to be a portion or a continuation of the lesion shown at *A* in Fig. 3. In other words, the third frontal gyre and the insula were neatly involved in the same circumscribed lesion, the second frontal convolution, the internal capsule, the basal ganglia, and all other portions of the brain not being the site of any old lesion. Although a section of the brain had not been made at the time of the publication of Dr. Gordinier's paper, his arguments in favor of a graphic centre based on a study of this case remain valid, as the second frontal convolution was not in the least involved.

I made many examinations of this case, and on a number of occasions used the patient at clinical lectures or demonstrations. The case was clearly one of aphasia with marked verbal amnesia and general disorganization of language on its dynamic side. Grammatical expression and combinations were lost. The patient could speak only in single words, or in short phrases, or very short sentences. What he said, however, he articulated and enunciated clearly; he had no paresis of the organs of articulation, enunciation or vocalization.

Besides the old lesion causing the aphasia the necropsy showed several recent hemorrhages—a clot in the left parietal region and two subpial extravasations, one covering the right occipital lobe and another of small size along the longitudinal fissure on the left side about three inches from the tip of the occipital lobe. A glance at the horizontal transection of the two hemispheres shows that the left hemicerebrum is much smaller than the right, the shrinkage appearing to be almost uniform for different regions of the brain. The ventricles and their horns are, however, not only relatively but absolutely larger than on the right side.

This patient, after an apoplectic seizure, developed aphasia, apparently without preceding paralysis or any other symptoms of

[1] Gordinier, H. C. Arguments in Favor of the Existence of a Separate Centre for Writing. THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES. September, 1903.

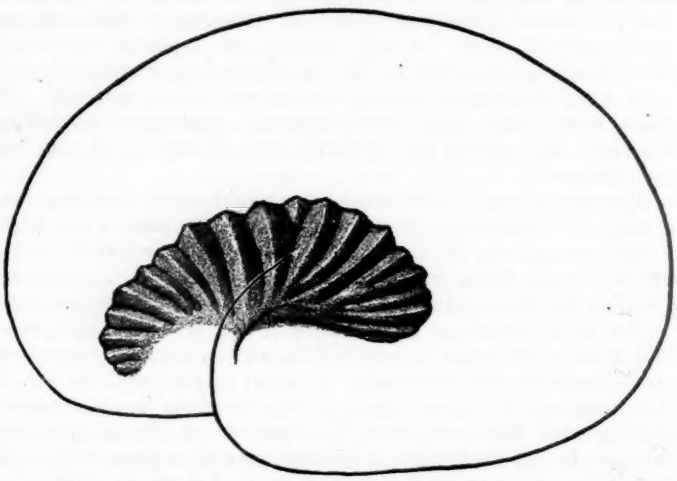
focal lesion like visual blindness and hemianopsia, When examined by me about nine years after the onset of the aphasia, he had almost complete inability to name persons and objects which he was able to recognize through all his special senses. He had also a marked form of paralexia. When he attempted to read, although he understood what he was reading, he repeated an absurd formula of a few phrases. He had limited spontaneous speech, even using short sentences without concrete nouns correctly. He could write many single words correctly, sometimes misspelling, however. He held his pen or pencil correctly and wrote with ease and firmness.

The part played by the insula in cerebral speech has long been a subject of discussion. In accordance with the plan of this paper I shall not go into the literature of the subject, but shall confine myself largely to an expression of personal opinion, giving the evidence for this opinion afforded by my own experience. Some of the most interesting data can be found in works on aphasia like those of Bateman and of Wyllie, and as regards recent literature, particular attention may be called to the paper by Dr. E. A. Spitzka on the "Redundancy of the Preinsula in the Brains of Distinguished Educated Men."[1] Some of the distinguished contributors to our knowledge of aphasia have even gone so far as to regard the insula as the sole cortical centre for speech. This view must, of course, be disregarded in the light of the numerous well-recorded cases of lesion of the subfrontal convolution with aphasia, and also when the numerous cases of degeneration or involution of Broca's convolution after lesion of the temporal lobe are considered. E. A. Spitzka holds that probably only the preinsula is concerned with speech, giving instructive personal observations and a review of the work of others in support of this position. His careful study of the brains of the Seguins, father and son, showed a redundancy of the left preinsula, also that the insulæ were marked by deeper fissures and a complementary plumpness of the gyres compared with ordinary brains. Exposure of the insula is usually regarded as evidence of disease or deficient cerebral organization, but Spitzka shows that this is not always the case, and gives what would appear to be the correct reason for the exceptions. While, on the one hand, deficient development of the opercula causes exposure of the insula, on the other hand in the highly intellectual, and especially in those with an unusual faculty of speech, a displacement and thrusting apart of the opercula also result in insular exposure. Spitzka gives some facts both from comparative anatomy and from an investigation of human brains in support of the view that the caudal portion of the insula has a different function from its cephalic portion. He gives, for instance,

[1] Medical Record, June 15, 1901.

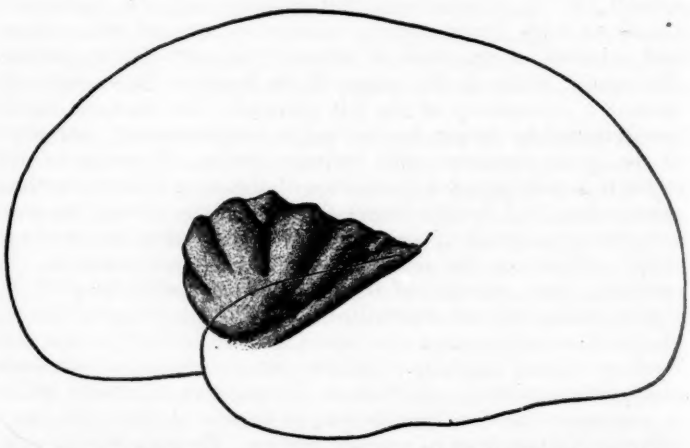
two illustrations, one (Fig. 5) a sketch of the insula of the bottle-nosed dolphin, the other (Fig. 6) of a normal well-developed human

FIG. 5.



Insula of the bottle-nosed dolphin. The caudal portion of the insula of this cetacean is more redundant than the cephalic. The entire insula is large, having, as shown in the diagram, as many as eighteen fissures and nineteen gyres. (E. A. Spitzka.)

FIG. 6.



A normal well-developed human insula, showing that relatively the cephalic is more developed than the caudal portion of the insula. The number of fissures and gyres, as will be seen, is less than one-half as great as that in the dolphin. (E. A. Spitzka.)

insula. The differences are striking. In the case of the dolphin the fissures number about twenty, while in that of the man they number five main fissures, two of these being subdivided. The gyres of the preinsular portion of the dolphin's insula are relatively small, while the reverse is the case in the human insula. It seems to me that much is in favor of the view that the preinsula is particularly concerned with speech. Up to the present time the students of cerebral morphology, anatomy, and physiology have not succeeded in locating with any certainty the cerebral terminus of the vestibular portion of the eighth nerve, although the writer has indicated it tentatively as probably being in the second frontal gyre. It may

FIG. 7.

Photograph of the insula of a man of average intelligence without brain disease.

be that the postinsula is related to the cortical centre of representation of the vestibular nerve as is the preinsula to the auditory centre. Spitzka regards the preinsula as an association area for speech, a cortical region which unites the sensory side of the zone of language or a portion of it with the subfrontal gyre, which he speaks of as the emissary speech centre. The writer's view is somewhat different, if he understands correctly the position of Spitzka. I believe that Broca's convolution and the insula (or perhaps only its anterior portion) constitute conjointly the same speech centre, a large cortical area being requisite for that portion of the mechanism of speech which is represented by this region. Neither of these areas has, I believe, projection fibres. Here language is organized for the use of the executive centres concerned

with speech, in other words, for the facial, lingual, and laryngeal centres situated anterior to the foot of the central fissure. This view is similar to that of Clozel de Boyer.[1] The specimens from the first case recorded in this paper (Figs. 1 and 2) support this contention, as when a circumscribed vascular lesion, either occlusion or hemorrhage, is limited to a certain area, it usually follows that the area affected has the same function. It is probable that the most important portion of the middle association area in the zone of language is situated in or near the midtemporal region, probably occupying portions of the second and third temporal gyres.

In one sense the insula and subfrontal gyre are undoubtedly association areas, but in them, to use the idea suggested by Broad-

FIG. 8.

Photograph showing the insula of a speechless idiot.

bent, language is organized into propositions. They constitute conjointly what might be termed a language-arrangement centre, but another association area concerned with the attachment of names to concrete concepts is probably situated in the temporal lobe.

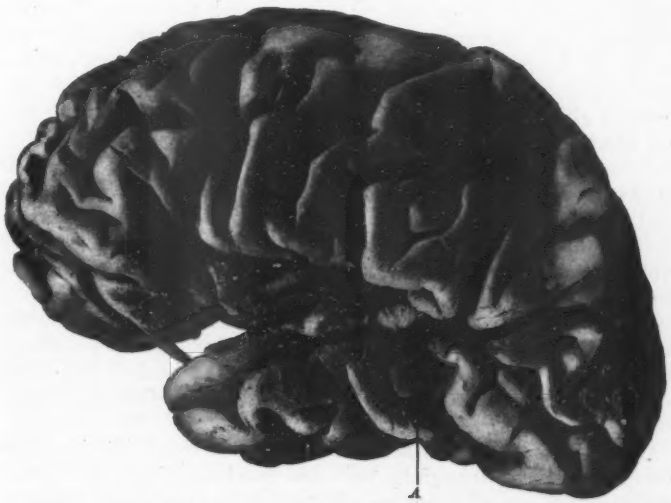
In Fig. 7 is shown a photograph of the insula of a man of average intelligence, but not well educated, the opercula being pulled aside in order to focus the island. In Fig. 8 is given a view of the insula of an idiot.

In Fig. 9 is exhibited the insula of a well-known case recorded in 1891 by the writer, which helped to fix the position of the centre

[1] Thèse de Paris, 1879, cited by E. A. Spitzka, Medical Record, June 15, 1901.

for word-hearing opposite the caudal extremity of the Sylvian fissure at the junction of the first and second temporal convolutions. The illustration shows extreme atrophy of the first temporal, retro-insular, and Broca's convolutions, with almost full exposure of the insula. In the light of what has already been said, these illustrations need but little comment. In the case of the man whose insula is shown in Fig. 7 no affection of language was present, the patient dying from spinal injury and its consequences; the idiot never acquired any power of speech, while the patient from whom the specimen represented in Fig. 9 was taken became word-deaf

FIG. 9.



Photograph of the insula of a case of lesion demonstrating the centre for word-hearing and the general auditory area. The original lesion was at *A*. The first temporal, retroinsular, and subfrontal gyres are much wasted. The first temporal convolution of the right hemisphere of this brain, with other parts, was also destroyed nine years before death by a second apoplectic attack, the first seizure causing the lesion at *A* having occurred fifteen years before death.

fifteen years before death, and nine years later totally deaf cerebrally because of a second destructive lesion of the right first temporal convolution and other parts.

The writer has long believed in the existence of a separate graphic or writing centre situated in the second frontal convolution, and has recently had the opportunity of obtaining new evidence of the most positive character supporting this view. Two classes of cases may be regarded as proving the existence of such a centre, namely, (1) cases in which a lesion absolutely circumscribed to Broca's convolution, or the insula, or both, causes aphasia without agraphia, and (2) cases in which a lesion circumscribed to the alleged position

of the graphic centre in the second frontal convolution causes agraphia without aphasia. I have had the opportunity of personally seeing two such cases. The case illustrated by Figs. 3 and 4 is, in fact, such a case, and I shall make no further reference to it, except to re-emphasize the statement that the patient was an aphasic but not an agraphic.

Recently in a case seen in consultation with Drs. J. W. McConnell, W. G. Spiller and C. H. Frazier, the existence of a graphic centre or motor graphic centre, as it is usually described, was positively demonstrated. The case had many other interesting features. It was one of tumor, largely prefrontal, removed successfully by Dr. Frazier at the Hospital of the University of Pennsylvania. I shall not go into details of the case, as I do not wish to forestall its fuller presentation by others, but shall, with their permission, call attention to the bearings of the case upon the question of the existence of a writing centre. The patient, a man twenty-nine years old, was first affected with a peculiar form of spasm attacking the face and head five years before coming under the observation of Dr. McConnell. Omitting all details of his history and the study of his case except in so far as it related to his agraphia and his mental condition, it may be stated that for over two years he had been changing in disposition and failing in intelligence. A portion of a letter written by him in 1902 shows some omissions of words and faults of spelling and construction, the same words in other parts of the letter being spelled correctly. These mistakes probably indicated the beginning of his agraphia. His recent letters were almost unintelligible. In consultation with Dr. McConnell I examined this man as regards his power to write, the day before the operation.

All attempts to write, either spontaneously or from dictation, were absolutely futile, although he held and handled his pen and pencil properly and copied a sentence with comparative ease. Illustrations of his efforts to write his own name and other expressions will be given in the full account of his case. The patient was not an aphasic. He had considerable difficulty in articulating and enunciating, particularly for a short time before the operation, but numerous tests showed that this was due to paresis of the organs concerned with articulation and enunciation, and was in no sense an aphasia such as results from a lesion of the subfrontal gyre and insula, such, in other words, as was exhibited by the patient from whom the specimens shown in Figs. 3 and 4 were taken. The tumor in this case lay athwart the second frontal convolution exactly in the position indicated in Figs. 1 at *G* and in Fig. 2 at the position marked writing. The reader interested in the subject of motor agraphia is referred to the papers of Gordinier.[1] One case

[1] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, September, 1903.

recorded by Gordinier is an almost perfect demonstration of the existence of the graphic centre.

Confusion still exists in the minds of many as to what is meant by the cerebral motor speech centre. Some, for instance, would make it include both the caudal portion of the third frontal gyre and the centres at the foot of the central or Rolandic fissure. As has already been indicated, the speech centre proper is circumscribed to the subfrontal convolution and insula. Here language is arranged for outward projection. Short association fibres connect this region with the true motor centres for articulation and enunciation, which constitute together the utterance centre or executive speech centre. Numerous physiological and clinicopathological facts and psychological principles could be readily adduced to uphold this view, but, following the plan of the paper as outlined, I shall content myself by referring to a single personal case and to one or two experimental observations. In 1889 I reported a case of paralysis strictly limited to the tongue, lips, and lower face, with slight implication of the palpebral orbicular muscle of the same side. Speech was entirely unaffected except as regards the effect on articulation caused by the lingual, oral, and slight facial paresis. The necropsy showed cortical softening involving the lower extremities of the central convolutions both on the lateral and Sylvian surfaces, and a spot one-half an inch in diameter about the middle of the internal portion of the insula. The softening reached into both the central and the precentral fissures. The anterior limit of the softening was one-fourth of an inch back of the anterior branch of the Sylvian fissure. This case seemed to clearly indicate that the true speech centre was confined to the subfrontal convolution and a portion of the insula, the area for the tongue, face, and allied movements being in the inferior portions of the central convolutions and largely in the precentral. In recent years I have come to believe that the motor region lies entirely or almost entirely in front of the central fissure. If there is any exception to this in the case of the human brain it is probably with regard to the centres for articulatory and allied movements. As is well known, the central fissure in the human cerebrum is only confluent with the Sylvian in extremely rare cases, and the entire gyral mass which separates its lower extremity from the Sylvian fossa may be concerned with movements which pertain to articulation, although by far the largest portion of the motor region, even in this position, is probably cephalad of the central fissure.

Grünbaum and Sherrington[1] found that unilateral faradization of Broca's convolution in the anthropoids evoked no movement, and in one case observed by the writer, faradization of the human

[1] Proceedings of the Royal Society of London, November 21, 1901; February 27, 1902, May 25, 1903.

subfrontal during an operation gave similar negative results, although in the same case the application of the electrode to the centres of the face, eyelids, and head gave prompt and positive response.

It is not necessary to record here cases of lesion of the area for the upper extremity (*WM*, Fig. 1), or its subdivisions, without involvement either of speech or writing. Such cases with necropsy have been seen by me, and a considerable number have been put on record. They simply show, as other cases referred to in this paper when discussing the writing centre, that the region of representation of the organization or arrangement of language for writing is independent of the area of representation of the movements of the upper extremity.

I shall make only brief reference to the auditory centre for speech, more commonly designated as the word-hearing centre. This is located at about the position *WH* in Fig. 1 and at that marked word-hearing in Fig. 2. As will be seen from a study of the diagram Fig. 2, the writer's present views with regard to auditory, visual, and concrete concept representation make it necessary to look upon some of the questions of speech localization, as of other forms of functional representation, somewhat differently from the manner in which these subjects have been hitherto regarded. In the cerebral cortex are situated primary auditory and primary visual centres, and, indeed, primary centres for all of the forms of sensibility, special and more general, as for cutaneous sensibility, smell, and taste. Each of these centres has surrounding it what may be termed its sphere of influence, this in the case of the auditory centre being a large area concerned with all forms of what in a broad sense might be termed object-hearing. Within this general region of special sounds-recognition are probably located a separate centre for natural sounds, like the notes of birds, another for musical notes, and still another for word-hearing. The centre of primary recognition is probably located, as represented in the diagrams Figs. 1 and 2, in the caudal portion of the first temporal gyre and in the retroinsular gyres. The word-hearing centre is on the outskirts of this region, at about the junction of the first and second temporal convolutions. It is not necessary to reproduce the evidence of this localization, which has been accumulating for years, and especially since Wernicke's first thorough presentation of the subject in 1874.[1] No more convincing case indicating the localization of this centre has been presented than that reported by the writer[2] in 1891. This case is now well known, and has been referred to in various articles and treatises. The illustration Fig. 9 is from a photograph of the left hemisphere of this case. From a study of the lesions

[1] Der aphasische Symptomcomplex, Breslau, 1874.
[2] University Medical Magazine, November, 1891.

found in this brain I concluded that the centre or area for word-hearing was situated in the caudal thirds of the first and second temporal convolutions, the focus of the area being about in a line with the extremity of the Sylvian fissure.

Built up by processes of education and through heredity, visual centres for numbers, letters, and words have developed out of a general area for object-seeing in that portion of the brain usually designated as the angular gyre (at the positions indicated by *WS*, *LS* and *NS*, Fig. 1, and by the terms word-seeing, letter-seeing, and number-seeing, Fig. 2). The evidence in favor of this highest visual area is of such a positive character and so well known that it is not necessary to adduce it in this paper. I would simply say that during the last two to three years I have had further corroboration of this localization in cases of brain lesion for which operations have been performed. In one of these cases the existence of a centre for object-recognition, independent of that for the recognition of words, letters and numbers, was indicated by attacks of transient mind-blindness, the lesion in this case probably occupying portions of the second, third, and perhaps of the fourth occipital convolutions. At the point marked *N* in Fig. 1, and naming in Fig. 2, is probably located a special region where names are correlated to the concrete ideas of objects which are recognized by sight, hearing, or other senses, or through the interaction of the centres of these senses. While the separate existence of this centre may be regarded as not absolutely settled, I believe the weight of evidence and of *a priori* considerations are in favor of its existence. From the nature of the work which it is supposed to do its situation should be related more closely to the sensory than to the motor side of the brain, and a portion of the third temporal convolution, or of this and a portion of the second temporal, as indicated in the diagrams, would seem to be its probable location. Some clinicopathological facts also point to this conclusion, as, for instance, a case described by the writer and McConnell, and others recorded by German observers. The reader interested in this subject is referred to the writer's articles on aphasia in his work on *Diseases of the Brain*,[1] and to a paper published in 1902.[2]

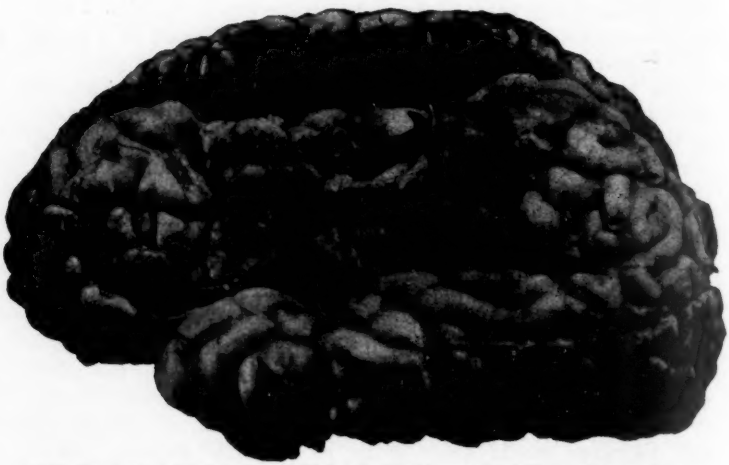
The correlation of the cerebral zone of speech with the sensory and motor regions of the brain is illustrated by those cases of mixed sensory and motor aphasia which are not infrequently observed. A common symptom-complex is that of nearly complete right-sided hemiplegia with correspondingly almost complete aphasia. Two or three such patients presenting this syndrome are usually to be found in the wards for nervous diseases of the Philadelphia Hospital—the hemiplegics with a single recurring

[1] The Nervous System and Its Diseases, Philadelphia, 1898.
[2] Journal of the American Medical Association, October 4, 1902.

utterance or with only one or two such utterances. The photograph of a specimen recently obtained from such a case is shown in Fig. 10. The case is identical in most respects with one reported by Burr.[1]

My patient was for many years in the Philadelphia Hospital, and was frequently used by me and others to illustrate lectures and demonstrations of aphasia. She was one of a group of cases studied by me with reference to pantomime as well as speech, and a clinical report of the case was made in Vol. II. of the *Philadelphia Hospital Reports*, published in 1893, the patient living eleven years after that time, but not changing in any essential particulars until her last

FIG. 10.



Photograph of the lateral aspect of the left hemicerebrum of a case of hemiplegia and complete aphasia, the apoplectic attack having occurred between fourteen and fifteen years before death. All parts of the brain supplied by the left medicerebral artery are necrosed.

illness. She was about fifty-one years old at the time of her death. Fourteen or fifteen years before her death she had an apoplectic seizure causing loss of power in arm, leg, and face, without aphasia; a few months later she had a second seizure, causing profound hemiplegia with contractures and aphasia. Sensation, so far as could be determined, was unimpaired. The deep reflexes in both upper and lower extremities were exaggerated on the paralyzed side. It was impossible to learn whether or not the patient was word-blind in the absence of her previous history. She was largely but not completely word-deaf. She was not mind-blind. "Her speech and pantomime," citing from the *Philadelphia Hospital*

[1] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, February, 1904.

Reports, "might be said to be summed up in her *la-la* and laugh and a much less frequent expression of anger or displeasure, which was usually accompanied by a movement of covering her mouth with her hand. The appearance of her face could not be said to be without expression, but, like her recurring utterance, it was an appearance nearly always the same. It might mean that she was pleased and it might signify assent or dissent; it might mean anything at all. Sometimes the *la-la* and the laugh became louder and more emphatic, but that was all. She never nodded the head in the usual manner for *yes* or *no*." This patient died in 1904, during the term of service of my colleague, Dr. C. W. Burr, to whom I am indebted for the opportunity of obtaining the results of the necropsy, the record of which will be omitted except as regards the brain.

Examination showed that the left medicerebral artery was occluded near its origin from the internal carotid, and necrosis of all parts supplied by the branches of this vessel had occurred. The first temporal convolution, the insula, the subfrontal, precentral, postcentral and inferior parietal convolutions were all necrosed and atrophied, as were also the ganglia and capsules and corona radiata, in so far as these are supplied by the branches of the medicerebral artery distributed to the interior of the brain. The left hemisphere was greatly reduced in bulk and had the appearance of being caved in between the prefrontal and occipital regions. The external appearances are shown, although not particularly well, in Fig. 10. The whole hemisphere is greatly reduced in size, as shown both externally and in a horizontal section made at the level of the insula. The parts necrosed included the entire motor region and its environing sensori concept motor speech zone, and in addition, large regions of the ganglia and capsules at the base. The lesions found in this case, as in that of Burr, are probably those usually present in cases of complete aphasia with a hemiplegia also nearly complete.

In some cases the aphasia is as nearly total as in the case just recorded, the hemiplegia being absent or represented only by an incomplete hemiparesis. In Fig. 5 is shown the photograph of the brain of such a case. This patient, like the one just recorded, was for many years a well-known character at the Philadelphia Hospital. Just as the other patient was frequently spoken of as *la-la*, so he came to be known as *anyone any*, this being his single recurring utterance. The history of the case is unfortunately lost, but the patient was well known to me, and was frequently used by me in clinical lectures and demonstrations. He was slightly paretic on the right side, but to so small a degree that he was quite useful as an assistant in one of the kitchens of the hospital, where he long worked. He was partially word-deaf. It is probable that he was completely word-deaf at first, and had to some extent recovered

word-hearing power. He was very acute in observing others when they were speaking, apparently sometimes recognizing what they said by the movements of articulation. To everything that was said to him he would simply answer *anyone any*, yet his appearance and manner sometimes indicated that he knew what was said, his knowledge evidently being obtained partly by sight and partly by hearing. His pantomime was not nearly as much disturbed as in the preceding case. Necropsy showed the conditions indicated in Fig. 11. The main trunk of the left medicerebral artery was not completely closed as in the preceding case, the branches occluded being those distributed to the insula and to the opercular convolutions, the third frontal, central, subparietal, and first temporal; those convolutions were so necrosed as to be almost obliterated.

FIG. 11.

Photograph of the left hemicerebrum of a case of complete aphasia with right hemiparesis. The subfrontal convolution, the insula, and the first temporal convolution and adjoining parts are completely destroyed.

It has seemed to me worth while to record the cases contained in this paper and at the same time to glance at the entire subject of the cerebral zone of speech, with the view of clearing up some disputed points and emphasizing others. The acquirement of speech is the first great step in the separation of man from animals lower in the scale. Man differs from the gorilla chiefly in his ability to talk and in being better looking. Relatively the brain of the gorilla and of the other anthropoids shows lack of development in the region given over in man to the zone of speech, while in these animals the primary centres for vision, audition, and movements are largely developed. The centres for word-hearing, word-seeing, for speech-associations and for writing have developed in the race and in the individual as these functions or attributes have been added to man's resources for communicating by language with his fellows. It is well that the students of comparative and of human anatomy and physiology, that clinicians and path-

ologists, should spend much time and labor in determining the existence and in accurately locating the various centres of representation in the zone of language, but the existence of these centres is ordained by evolution. The position, the size, and probably the shape of the cortical areas which are spoken of as the centres concerned with speech are determined by unalterable biological principles. The anthropoid and the human savage alike have large cerebral areas for object-recognition, whether through sight, hearing, touch, or the other senses. As man learns to talk the auditory speech area or centre develops in close proximity to the regions of primary representation of sound. Similarly, centres for visual speech develop within the sphere of influence of the primary visual centre, and the areas of language arrangement both for speech and writing in proximity to the great motor area, parts of which become the final cerebral instruments of speech. It is just as necessary that the man who writes spontaneously should have a centre for language arrangement for writing as that he should have a similar centre for speech, and the same is true of every subarea so far determined in the great zone of language. With our present knowledge of cerebral structure and function it is just as certain that a separate graphic centre or a separate centre for word-hearing or for word-seeing has its place in the human cortex on the principles here enunciated, as appears from the records of convincing clinicopathological cases by Broca, Gordinier, Eskridge, Dejerine, Wernicke, or anyone else.

TRAUMATIC HEMORRHAGE OVER THE THIRD ANTERIOR FRONTAL CONVOLUTION; OPERATION; REMOVAL; RECOVERY; REMARKS ON PRESENT STATUS OF BRAIN SURGERY.[1]

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The surgical invasion of the brain begun so auspiciously under the leadership of the English surgeons Horsley and MacEwen, has not proven so successful as the first reports seemed to indicate; nevertheless immense strides have been taken in this direction, and beneficial results have been obtained in a once barren and hopeless field.

[1] Read at the Ninety-eighth Annual Meeting of the Medical Society of the State of New York, January 26, 1904.

Tumor, abscess, hemorrhage, epilepsy, microcephalus, insanity, and the removal of the Gasserian ganglion for tic douloureux are the indications most frequently calling for surgical intervention, and the trephine has become an all-important agent in the treatment of these various lesions. The success in any of these conditions depends upon (1) an accurate diagnosis made early in the course of the disease; (2) an accessible lesion demanding the minimum of brain destruction; (3) a faultless surgical technique.

With these conditions anticipated and overcome, results will follow which will restore to brain surgery much that has been lost by indiscriminate surgical meddling which marked the early period of this procedure. Up to January 1, 1903, Starr[1] found in medical literature 365 cases of brain tumor operated on, 315 being cerebral and 50 cerebellar, with the following results:

	Total.	Cerebral.	Cerebellar.
Cases in which the tumor was *not* found	111	91	20
Cases in which the tumor was found, but *not removed*	27	21	6
Cases in which the tumor was removed and the patient *died*	59	51	8
Cases in which the tumor was removed and the patient *recovered*	168	152	16
Percentage of failures	38		
Percentage of deaths	16		
Percentage of recoveries	46		

These figures are sufficient to give confidence and to stimulate further efforts in this direction.

Woolsey,[2] in an instructive paper on the surgery of cerebral tumors, including a table of 101 cases of cerebral tumors, emphasizes the following conclusions: 1. The sphere of operation for cerebral tumors may be and has been extended to those parts of the cortex where tumors are accessible and localizable—*i. e.*, to the prefrontal, parietal, and occipital regions, in addition to the motor area. 2. The prognosis, both immediate and remote, is as good or even better than in operations for malignant growths in some other locations. 3. This prognosis has improved with the improvements in localization and operative technique, and with the limitation of the radical operation to cases accurately localized. 4. The palliative operation is strongly indicated to relieve symptoms where localization cannot be accurately made or the tumor cannot be removed. The exploratory operation is contraindicated. 5. Practically all circumscribed growths of moderate size are suitable for operation. 6. The osteoplastic method should be employed and the most rapid and perfect technique adopted which the circumstances allow.

The application of surgery for the relief of traumatic insanity and traumatic epilepsy has not been as favorable as anticipated,

[1] Journal of Nervous and Mental Disease, July, 1903, p. 398.

[2] The American Journal of the Medical Sciences, vol. cxxvi. p. 956.

and operations for these conditions are becoming justly less and less. In 33 cases of epilepsy operated on at the Craig Colony, Sonyea, N. Y., Dr. Spratling[1] noted the following results:

In 21 there was no improvement in the disease, either temporary or permanent. In 8 the attacks were lessened in frequency and severity, the operation being a part of the treatment only. In 3 the disease was much worse after the operation. In 1 there was apparent recovery.

The writer reported a case of traumatic epilepsy[2] operated upon May 2, 1891, with freedom from attacks for twelve months, then recurrence. A second operation on October 15, 1892, with freedom from attacks for three years; a third operation was performed in 1895, with favorable results for three years, and finally the attacks becoming again so frequent a fourth operation was carried out November 25, 1902, and the patient died from the shock. The opening in the skull measured four inches in diameter. In idiocy and imbecility with microcephalus, brain surgery has been most disappointing. Starting with the enthusiastic report of Lannelongue in 1891, the early reports looked favorable. As time went on the successes were less frequent and to-day the operation is rarely performed except where there is simple development and not where extensive lesions exist (Dana).

Spratling[3] presents 194 such cases in the paper referred to under two heads in condensed form, the first group including the more immediate results in 111 cases, with the following results:

Died in consequence of operation or soon after	19 or 17 per cent.
Operated on with no result	25 or 22.5 "
Operated on with slight result, but not satisfactory	10 or 9 "
Improved in stated ways	24 or 21.5 "
Improvement without reports as to its character	30 or 27 "
No reports as to results given in general	3 or 3 "

The second group of 83 cases with results somewhat more permanent was as follows:

Died	20 or 24 per cent.
Unimproved	54 or 65 "
Improved	9 or 10⅓ "

Lowenstein voices, perhaps, the popular notion regarding this operation: "With the hypothesis of the primary synostosis of the sutures and fontanelles and the secondary hindrance of brain development stands or falls the right of Lannelongue's operation. The hypothesis is false and therefore the operation is not a suitable one. Death or no results follow."

The Krause-Hartley operation for the removal or breaking up of the Gasserian ganglion in cases of obstinate facial neuralgia

[1] American Journal of Insanity, July, 1903, p. 27.
[2] Buffalo Medical Journal, November, 1895.
[3] Loc. cit.

has been measurably successful and continues to receive attention. Two cases recently presented before the Buffalo Academy of Medicine by Drs. Marcel Hartwig and William C. Phelps were entirely relieved of their torture by this operation.

Perhaps the most favorable field for the trephine is in cases of subdural hemorrhages due to traumatism or to spontaneous ruptures during violent spasmodic seizures, as in whooping-cough. These hemorrhages, whether extradural, subdural, or cortical, fall within the jurisdiction of the surgeon, and as a rule, if operated upon early, give very gratifying results. Whether or not these hemorrhages would absorb without operation depends upon the extent and source of the hemorrhage; the surgeon no doubt in some cases does no more than hasten what would naturally have occurred.

Success in cases of hemorrhage, as in brain tumors, also depends upon an early diagnosis, a correct localization, and a faultless surgical technique. The following case of *contre-coup* hemorrhage due to traumatism is deserving of publication, and resembles in some respects the case reported by Starr in *Brain Surgery*, 1893, p. 161. As in that case, so in the one to be reported, a complete recovery followed the removal of the clot.

L. B., male, aged sixty-two years; height, five feet eight inches; weight before accident, two hundred and two pounds; constitution strong, vigorous; heavily built man. He had always enjoyed the best of health, had never contracted syphilis, but for years had been moderately addicted to the use of liquor.

On Monday evening, August 10, 1903, at about 7.30 o'clock, while trying to pass from the rear platform of an open trolley car along the running board to a seat, he stumbled against the wheel guard and was precipitated to the pavement. The car was running at a speed of perhaps five miles per hour, and he struck the asphalt pavement on the right side of the head, over the region of the right eye, and in trying to save himself struck on his right shoulder and right hand. As a result of the fall he was somewhat stunned, but declared to Dr. Pfohl, who was on the car, that he was "all right" and not injured to any extent. He was driven to his home accompanied by the doctor, who found the following condition:

The right eye was greatly swollen and discolored; the upper eyelid stuck out like a cup; the conjunctiva was greatly ecchymosed, overlapping the cornea, and the eye was practically closed by the effusion. The right shoulder was bruised and discolored, and one of the metacarpal bones of the right hand was fractured. He complained of great pain over the occiput during the night, of such intensity that the neighbors were disturbed by his cries and groans. He had eaten a hearty supper that evening and vomited at intervals throughout the night.

The following day, Tuesday, he was more or less under the influence of narcotics, but vomited occasionally. He conversed

with his wife and daughters on various topics and appeared to be recovering rapidly from the fall. His son, a careful observer, visited him at noon on Tuesday, and with the exception of the discoloration noticed nothing wrong about him. He talked about the accident and business matters, and when asked if his employés should be notified said, "No, I will be out in a few days." At 11 o'clock that night he was sitting up with his family, reading the newspaper and engaged in general conversation. He slept fairly well Tuesday night.

On Wednesday morning, August 12th, at 6 o'clock, his wife approached his bed and asked if there was anything he wanted, to which he made an unintelligible reply. Thinking he was jesting his wife asked, "What language are you talking, Italian or Polish?" He evidently did not recognize her, but kept on jabbering in an unintelligible and incoherent manner. To his sister, who visited him this morning, he said, "Hello, Clara!" very plainly, then jabbered for some seconds.

Wednesday noon his son called again, and to the question how he felt replied, quite distinctly, "All right." On attempting further conversation with the father the son noticed that the first two or three words of a sentence would be distinctly spoken and the remaining words would be an unintelligible jumble, as if spoken in a strange language. As time went on the distinctly spoken words became less and less and conversation was stopped. During the day he grew more confused; became partially unmanageable; insisted on going to the toilet room; refused all nourishment; left the bed several times, and tried to clothe himself. Being a strong, powerful man, he was unopposed in his desires and actions. He did not complain of any headache and had not vomited this day. The case having assumed a more serious aspect, Dr. Marcel Hartwig was called into consultation. The following day, Thursday, August 13th, I was asked to meet Drs. Pfohl and Hartwig in consultation.

The patient was in bed, but during my stay at the house arose twice and, slightly assisted, went to the toilet room at the other end of the house. He did not show any peculiarity of gait, did not stagger nor go sideways, and did not fatigue perceptibly by the exertion. He would not use the bed-pan, and although recognizing the danger of further hemorrhage, still we did not deem it wise to force matters with him.

His face was badly disfigured and discolored, especially about the region of the right orbit. He did not seem to know me or to appreciate his surroundings. He was stuporous, breathing heavily, his cheeks ballooning in expiration, and only with difficulty was his attention riveted for a few moments. He did not reply to questions put to him, and only once did he mutter something in reply to Dr. Hartwig.

There was no paralysis of the facial muscles, or of the arms or legs; neither was there any tremor or twitching of any of these muscles. Pupils equal and react to light. The tendon reflexes and deep reflexes were normal, and no disturbance of sensation.

The pulse was 72, temperature 98.5° F., and respiration 20. The following day, Friday, found him in practically the same condition, perhaps somewhat more stuporous and a temperature of $100\frac{4}{5}$° F. in the morning and $101\frac{3}{5}$° F. at 6.30 P.M. He drank milk and water freely, and did not complain of headache. Early in the evening his son noticed a slight tremor of the chin and later on the family observed that at frequent intervals he would without moving his head slowly turn his eyes toward the right. They thinking he was annoyed by a picture on the wall removed it, but it had no effect on the patient. At times his lips trembled and he uttered a cry like "mum, mum, mum." The nurse had been instructed to watch the right arm carefully for twitchings or contractions and to report at once their appearance.

At 10.40 Saturday morning the nurse noticed that the patient would have convulsive seizures of the right side of the face instead of the arm, becoming more and more frequent, and at once telephoned for the physicians. Arriving at the house I found Drs. Hartwig and Pfohl, who had been eye-witnesses to several of these attacks, and it was not long before another one made its appearance.

The patient was lying on his back quietly, then slowly moved his head slightly toward the right. The eyeballs in turn moved toward the right and then the eyelids were closed. This spasmodic action lasted but a few seconds and was followed by a series of clonic convulsions. The forehead was not involved except by transmission. The whole right face moved up and down convulsively; the mouth was drawn to the right, and the jaw worked in unison with the face. A peculiar sound was made by the working of the lips, as if smacking, and the tongue could be seen between the teeth jerking and pointing toward the right. The right pupil was dilated, but as the left eye had been atropinized the day before no comparison of the two pupils could be made.

Following the clonic seizure, which lasted perhaps thirty seconds, the patient resumed his normal appearance and breathed heavily for a few moments. There was no involvement of the right arm or leg, no foaming at the mouth, and no involuntary urination.

It was now definitely decided to urge upon the family the necessity of an immediate operation. Consent having been willingly given, it was decided to operate at 3 o'clock that afternoon. While this conversation was going on in the sick-room, unfortunately the patient overheard and understood its full import, as he was seen to cry, and sobbed for some time. He assisted in dressing himself, and on looking out of the window and seeing the ambulance before the house, said very plainly, and distinctly, "No hospital for me."

He was greatly agitated and aroused and fought all efforts to go. A carriage was thereupon ordered and it was thought he might be induced to enter under the guise of going for a drive; but he waved his hand and said again, very distinctly, but pitifully, so that all in his vicinity could hear him, "You can't fool me." He was then forcibly picked up, carried into the ambulance, and hurriedly driven to the German Hospital.

Surgical Report by Dr. Marcel Hartwig.

Concerning the surgical features of this interesting case, which is the second in my experience, there is not much to be said. *Contrecoup* bleedings are frequently due to ruptures of the arteria meningea media or its branches. In this case I could not establish this fact, although the quantity of blood clots between pia and dura amounted to about three teaspoonfuls, and the flow of subdermal blood was considerable. We have, therefore, to conclude that some venæ communicantes were torn, the surface of the brain itself looking too good in the trephine opening to believe that a rupture of the brain itself should have existed in the covered neighborhood. Drainage by two catgut loops, one forward and one backward, inserted respectively one and two inches deep, was sufficient to take care of the leakage after the trephining. A fissure in the bone was not discovered. Healing under the first dressing. The dura was partly united by catgut. The thinned catgut strands were withdrawn on the fifth day. Two convulsive seizures of light type occurred the first night, not to appear again. The skilled assistance of Dr. H. J. Waterman, of New York, was my pleasant privilege to have on this occasion.

Following the operation he had no spasm until 10.50 P.M., when the usual convulsive seizure appeared. This was followed by another seizure at 12.30 A.M. and 1.25 A.M. Sunday. His pulse during the night ranged from 72 to 90, temperature from 103° to 104° F. He muttered unintelligibly during the night, was restless, passed urine involuntarily, and tried to tear off bandages. During the night from Sunday to Monday he uttered many intelligible words, as, "I must go down to the store," and concerning business matters. He made many motions about giving out and receiving money; imagined he was smoking, going through the motions of putting the right hand to his lips and then knocking off the ashes from cigars. The hospital physician noticed some twitching and blinking of the right eye, with some involvement of the face and chin of the right side. These attacks were very slight and lasted only a few seconds. These light seizures, it may here be stated, were the last to be observed, occurring about forty-eight hours after the operation.

Pulse on Monday, August 17th, ranged from 76 to 88, temperature from 102° to 103° F. On Tuesday, the 18th, he spoke at times very plainly, calling for his children and talking at random about his business affairs.

Wednesday, August 19th, the dressings were changed by Dr. Hartwig and showed slight staining with cerebrospinal fluid. No further hemorrhage had taken place. The wound was healing rapidly by first intention, and with the exception of a semistuporous condition at times, the patient was progressing satisfactorily. On being roused he answered questions plainly. For instance he said, "I want a drink of cold water," "I am tired." He asked what time it was, and when told it was 8 o'clock, said, "I want to go to breakfast." Pulse to-day ranged from 80 to 94, temperature from 101° to 101⅗° F.

On Thursday, August 20th, he gave his name and address correctly, spelling his name, and spoke quite audibly on being questioned. A mild delirium comes over him frequently during the day, characterized by incoherent language about his business affairs, tearing off bandages, and escaping into the corridors. At night he imagines people looking in through the window. Thinks he is under arrest and offers to settle. He becomes very restless and is with difficulty quieted.

On August 27th the wound had thoroughly healed, the temperature had fallen to 99° F., pulse 72 to 75, and urination ceased to be involuntary. A decided general improvement had taken place in his general condition, the delirium had passed away, and all irregularity of speech had ceased.

On September 2d, 4.30 P.M., the nurse reported as follows: "For the first time as the newspaper was lying on the sofa, the patient reached out and took it. He opened it and read aloud from the first page as correctly and as good as one could ask without glasses."

He wrote some short letters on September 5th, and on the 9th accompanied his wife to the street car.

On September 12th he was dismissed—cured. His urine during his stay at the hospital contained a small amount of albumin.

September 15, 1903, accompanied by his nurse, he reported at my office, when I made the following examination: The flap on the left side of the head has healed perfectly. It is two inches wide at the base and two inches long. The rounded apex is three and a quarter inches above the external auditory meatus. The distance from the upper incision to the external auditory meatus is three and three-eighths inches. The pupils are equal, react to light and accommodation; the fundi are normal; papillæ not inflamed. The two sides of the face are symmetrical and functionate equally; tongue when protruded shows no deviation and the uvula hangs straight. There is no perceptible change in the character or volume of his voice, and he says that friends to-day recognized his voice over

the telephone without knowing beforehand who was at the phone. There is no difference between the sensation of both facial halves. A mental peculiarity still present is his inability to remember names. He forgets name of nurse, of the firms with whom he transacts business, and of old acquaintances. He complains of pain over and about the seat of the wound, commencing over the left eye and travelling backward beyond the scar. He says that before the operation he was very near-sighted, now is much better in regard to vision, and can see to read names across the street. The nurse

FIG. 1. FIG. 2.

FIG. 1.—Showing the outlines of scalp wound and depression of skull caused by trephine opening, one month after operation.

FIG. 2.—Showing trephine opening and underlying brain convolutions.

was retained for two weeks, during which time the patient's memory returned to its normal, and he began his ordinary routine work the forepart of October, 1903.[1]

The case is a remarkable one in many respects. That a basilar fracture of the skull did not follow the heavy blow of his head on the pavement is unusual. Severe concussion of the brain did not even follow, as he was not rendered unconscious, and said after being picked up, "Oh, I am not hurt much." The pain and attacks of vomiting the following day were the only evidence of any cerebral

[1] June 15, 1904, patient is in every respect in same good health as before the accident.

injury, and were it not for his contused eye and face he would have been at his business on Tuesday, August 11th. What happened was a small localized hemorrhage of the brain membranes on the side of the head opposite to the infliction of the blow, commonly known as *contre-coup* hemorrhage. This occurred immediately over the cap of Broca, third anterior frontal convolution of left hemisphere. The hemorrhage was slowly progressive, as no difficulty of speech was noticed until some sixty hours after the injury, producing symptoms of aphasia and paraphasia. The extension of the hemorrhage backward and downward, involving the facial centre, was no doubt due to the gravity owing to patient's recumbent position. The vessel injured was undoubtedly a small vein of the pia, situated perhaps just anterior to the speech centre, and not producing localizing symptoms until involvement by pressure of the neighboring brain cortex. The slowly on-coming procession of symptoms would vouch for this conclusion, as headache and vomiting the second day, aphasia and paraphasia the third day, and facial epilepsy of the Jacksonian type on the fourth day after the injury. Whether the arm and other centre would have become involved later is questionable. As the dressings on the fourth day after the operation showed no blood stains, only those of cerebrospinal fluid, it is safe to assume that the pressure of the clot upon the injured vessel was sufficient to stay further hemorrhage, and that no other centres were liable to compression. The diagnosis of hysteria was not to be totally disregarded.

The tardy appearance of symptoms after a railway injury, peculiar behavior of patient, his inability to converse or even to utter intelligible words from Wednesday night until he saw the ambulance in front of door, and then distinctly saying, "No hospital for me," and "You can't fool me," could all be easily regarded with suspicion, and the diagnosis of hysteria was never wholly lost sight of until the appearance of the facial epilepsy of the right side. This fact, coupled with the aphasia, showing successive involvement of neighboring brain centres of the left hemisphere, was absolutely conclusive and made the diagnosis unconditional. The finding of the clot with small loss of the calvarium, its speedy removal, the hasty healing of the wound, absence of all sepsis, and complete restoration of the patient to perfect health, all within a few weeks, show surgical skill of marked degree.

THE ADAMS-STOKES SYMPTOM-COMPLEX, WITH REPORT OF A CASE.[1]

By Clarence Quinan, M.D.,
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In 1893 Huchard proposed the name Adams-Stokes disease for a remarkable symptom-complex characterized by temporary or permanent bradycardia, loss of consciousness, together with apoplectic or epileptiform seizures. According to him the condition is usually if not exclusively encountered in individuals of advanced years, and is indicative of arteriosclerosis of the cardiobulbar type.

The first authentic case was reported by Adams in 1827, but those of Stokes, published in the early fifties, were better described and have been more frequently cited. A long interval elapsed before the observations of these earlier writers were accorded proper recognition, though other clinicians reported cases from time to time that were similar in every essential point. Among the more important contributions of this period are the papers of Cornil (1875) and Handfield Jones (1876). Both of these authors accurately outlined the peculiar symptom group and described cases that undoubtedly must be included in the same category with those of Adams and Stokes.

It was not until after Huchard had suggested a definite pathological basis for the complex that it was submitted to critical analysis, and some years passed before it was accepted as a distinct clinical entity. Case reports of more or less interest appeared in considerable numbers at this time, and we find casual mention of the condition in the text-books of Balfour (1894) and Edgren (1898). In 1899 a valuable paper was published by His in which he described for the first time clinically the unusual complication known as blocked heart or atrioventricular allorhythmia. In 1901 August Hoffmann considered the neuroses of the heart and bloodvessels of sufficient importance to justify separate consideration. His treatise on the subject was shortly followed by those of Cassirer and Ludwig Krehl. We are indebted to these authors for the systematic presentation of the neuroses of the heart, including the Adams-Stokes complex; however, it is necessary to supplement their statements by reference to the very able reports that have appeared since, especially those of Luce (1902), Jaquet (1902), Lewy (1902), and Osler (1903). A fair idea of the general literature of the subject may be obtained from the painstaking review recently compiled by Edes (1901).

[1] Read before the California Academy of Medicine, March 22, 1904.

It is proper to call attention at this point to the important services rendered in the development of functional heart diagnosis by von Basch, to whom we owe the sphygmomanometer. Mention should also be made of Schott, Gräupner, and of A. Smith, who have done much to perfect mechanical methods for the determination of the driving energy of the heart, and who have thereby aided materially in the investigation of the neuroses.

ETIOLOGY. A pulse rate under sixty is usually considered pathological, though Huchard states in his classification of the bradycardias that it is normally slow in some individuals. Napoleon is said to have had a pulse rate of 40, but there is reason to believe, from the statements of Corvisart, that he was subject at times to peculiar seizures which have never been fully described. Bradycardia may be induced experimentally by irritation of the pneumogastric nerve, compression of the carotids, or by a rise of arterial pressure. It may at times be observed after childbirth and during the period of convalescence from acute articular rheumatism and a number of the infections. Schuster (1896), for example, describes a case in a child of four after an attack of acute articular rheumatism, with a pulse rate varying from 35 to 130. The respiration was slow and irregular, and there were profuse sweats after each period of bradycardia. The case was probably one of acute myocarditis. Bradycardia may be caused by certain of the inorganic or organic poisons, and not infrequently is present in certain autointoxications, as, for example, in retention of bile salts without icterus (Brandenberg, 1903), and in the course of a number of morbid gastrointestinal hydrolyses. Slow pulse may occur in neurasthenia, hysteria, epilepsy, melancholia, amyotrophic lateral sclerosis, and tabes, but in none of these diverse conditions is the true clinical picture of the Adams-Stokes disease fully developed.

The Adams-Stokes complex most commonly occurs after middle life. It is rarely seen before the third decade. Of the twenty-five cases that I have analyzed, 76 per cent. were over fifty years of age. Males are more frequently affected than females, the ratio being nearly three to one. Syphilis may be a contributing factor, but was present in only two cases. Curtin (1903) has noted bradycardia with convulsions after excessive use of tobacco. Alcohol appears to have a definite causal relation. In a few instances a life of exposure seemed to predispose, but this factor cannot be emphasized. In the cases of Balfour and von Schrötter traumatism seemingly played a part. The typical complex may be associated occasionally with sclerosis of the bulbar vessels and attendant anæmia of the vagus centre, or parenchymatous changes may be found in the peripheral filaments of this nerve. Abortive forms are met with in individuals of neuropathic tendency. In this group belong the peculiar attacks of temporary bradycardia with dizziness, partial loss of consciousness, or of transient weakness of the extrem-

ities sometimes seen in neurasthenic or hysterical patients after periods of emotional excitement. These cases, as a rule, have a labile circulation and yield definite evidence of vasomotor instability.

Morbid Anatomy. The post-mortem findings are inconstant. Anatomical lesions of the heart and bloodvessels are relatively frequent. Artery degeneration may be widespread, but it is more common to find regional sclerosis. The fallacies that beset conclusions based upon macroscopic observation alone are clearly shown by the very thorough studies of Luce (1902). This author noted acute diffuse parenchymatous vagus neuritis with total disappearance of the axis-cylinder processes at many points, although at autopsy both vagi were regarded as normal, and nothing pathological was found in the medulla oblongata or the vagus centres. The arteries at the base of the brain sometimes present gross degenerative changes, but this is rather the exception than the rule. In the absence of accurate histological data, the greater part of the pathological reports at present available must be regarded with distrust. Undoubtedly in many instances essential changes in the peripheral vagi have been entirely overlooked.

Among the conditions affecting the heart itself those due to interference with its blood supply are most important. Sclerotic changes in the coronary vessels are frequently though by no means invariably present. Partial occlusion of one or both vessels may occur as the result of calcareous deposits at their points of origin. In one case, that of Sendler (1892), the vessel was partially occluded by the pressure of a primary fibroma of the heart.

In uncomplicated cases the heart is often not enlarged. Fragmentation of the muscle of the left ventricle has been reported, but in every instance must be regarded as an artefact. Fatty degeneration of the heart is not constantly present, as Friedreich thought, though it is common to find areas of fatty or fibrous myocarditis. The valves are often found intact in cases that had pronounced murmurs during life and gave every evidence clinically of extensive valvular disease. There may be imperfect alignment of the aortic segments, however, due to the presence of sclerotic nodules. In the remarkable case reported by Luce there was a primary round-celled sarcoma of the heart of the size of a hazelnut situated in the right ventricle, which partially invaded both the aortic and pulmonic orifices and gave rise to true bilateral obturating stenosis. As a rule there are no characteristic changes in the abdominal organs. The kidneys may be quite normal. Hoffmann (1901) quotes two cases of exceptional interest: that of Holberton, in which there were found narrowing of the occipital foramina, together with parenchymatous changes in the vagi and the superior cervical ganglion, and Lépine's case, due to compression of the pons and medulla.

SYMPTOMS. Bradycardia, either permanent or recurrent, is always present, accompanied at irregular intervals by central nervous manifestations that are characteristic. The typical seizure may be preceded by a definite aura: sensations of weight and uneasiness in the epigastric region, or a sudden rush of blood to the head or extremities, are the more common. Stokes' patient was able to avert an impending attack by dropping on his hands and knees and holding his head down. Balfour mentions a parallel instance. His patient was able on one occasion to accomplish the same purpose while out walking by lying down in the road. In the fully developed attack there is sudden loss of consciousness lasting from a few seconds to hours, but usually of momentary duration. The patient falls; convulsive movements of the extremities ensue; the pupils are dilated; the respirations stertorous and at times of the Cheyne-Stokes type. The pulse rate is invariably diminished, and counts of 20 and under are not uncommon. In one case a rate of 5 was noted. There may even be gradual extinction of the radial pulse, with temporary cessation of heart action. In my case no pulse could be detected at the wrist during the attack. Various authorities claim to have heard indistinct tones or murmurs over the præcordia in the period of suspended activity. Hoffmann states that with the radiograph he was able to detect movements of the auricles while the ventricles were at rest. All attempts on my part to verify this observation were wholly fruitless owing to the impossibility of controlling the position of the patient during the convulsive phase.

The initial attack is usually of sudden onset and may affect an individual apparently in good health. In other instances there is an antecedent history of occasional dizzy spells extending over a considerable period. These prodromal attacks are, as a rule, of transitory nature and cause no particular inconvenience or apprehension. Once established, they tend to increase in frequency, though well-directed treatment will often give relief and even comparative immunity for some time. The attacks are subject to extraordinary variations both as regards frequency and severity. There may be several in the course of a day and then none for a week or more. Again, the seizures may be of hourly occurrence and lead rapidly to a fatal issue. I have frequently seen five or more in the course of an hour. His's patient had 143 in a single day. The exciting causes are indefinite, but it is certain that psychical factors play a much more important part than is generally stated. As a rule these patients are profoundly responsive to changes of environment, and under stress of emotional excitement may have frequently repeated seizures. In the milder attacks consciousness may be partially preserved and the patient able to maintain his balance by grasping near-by objects. In others loss of consciousness is absolute. Muscle movements set in, varying in intensity from

fibrillary twitchings to the most powerful convulsions, and the patient finally recovers consciousness to find himself in the prone position with no definite idea of what has happened. As a rule there are no serious after-effects, and the general health in the free period is unimpaired.

Evidence of vasomotor unrest is seldom lacking. Extreme pallor of the face, alternate blanching or reddening, coldness of the extremities, and drenching sweats after the attacks are the most common signs. It is difficult to escape the conviction that vasomotor disturbances initiate the attack in a large number of cases. Both Ortner (1903) and Jaquet emphasize this point. This is less true of those cases that exhibit permanent vagus overaction than of the larger group of recurrent bradycardias which usually are of labile type and frequently have pulse rates above normal in the interval between attacks. It is in this latter class that the vasomotor influence is the more marked.

Certain anomalies of the circulation are of special interest:

1. *Positive Centrifugal Pulsations of the Jugular Vein Exceeding in Frequency the Apex Beat.* This remarkable phenomenon, the clinical description of which we owe to His, appears to be due to auriculoventricular asynchronism or allorhythmia. As Sahli has pointed out, if we accept the evidence yielded by the experiment of Stannius, namely, that the idiomuscular movements of the heart arise in the auricle and are propagated thence to the ventricle, and that it is possible to produce dissociated movements by interrupting the path of the impulse at the auriculoventricular border, it follows that a lesion at this point may cause independent auricular contractions, with no accompanying movements of the ventricle. This affords a logical explanation of the venous pulse observed by His. His' patient had a radial pulse rate of 18 to 32 exactly isochronous with the apex beat, while the jugular pulse ranged from 96 to 104 and was isochronous, with a low, humming murmur heard to the right and left of the sternum in the second and third intercostal spaces. One of Stokes' patients (Edmund Butler) affords a parallel example, though the phenomenon was less well marked. Blocked heart must be regarded as of very grave import. According to Belski (1902) it is peculiar to the terminal period of various cardiopathies and indicative of a dying myocardium. For reasons that are sufficiently obvious, the venous pulsations observed in blocked heart may be confined to the bulbus jugularis.

2. *Heart Bigeminism or Pseudohemisystole.* Hoffmann observed this form of arrhythmia in a young woman who was subject to syncopal attacks and had a pulse rate varying between 18 and 30. According to Dehio it is possible to orient the causative lesion by the action of atropine. This drug will equalize the pulse rates at the heart and periphery if the vagus centre is involved, but not if the bigeminism is due to mural changes in the heart.

Special Physical Signs. 1. *Heart.* Inspection may reveal little of objective value. The rib cage usually is stiff and its range of movement restricted. There is no bulging in the precordial region. In a majority of patients the apex impulse is very faint or invisible and frequently cannot be detected in any position. As a rule it is within the nipple line.

Clinical and pathological reports concerning the size of the heart are not in harmony. Very commonly there is no demonstrable enlargement. Moderate hypertrophy of the left ventricle occurs in the cases of diffuse arteriosclerosis, and dilatation of the right ventricle usually takes place as a terminal event.

The heart sounds may be normal both at the apex and base. In some patients they are faint and distinguishable with difficulty; in others the tones are loud and ringing, with accentuation of the second aortic sound. Murmurs are relatively frequent. A soft blowing systolic murmur at the apex is the most common, but occasionally loud systolic murmurs are heard at every orifice, and it is astonishing over what a large area these sounds may be audible. Coincident pulmonary stiffness and distention, to which von Basch and, later, Zuelzer (1901) called attention, may facilitate the diffusion of the sounds by offering a better vibratory medium. Alien tones of doubtful genesis occasionally invade the heart cycle. They are usually low and indistinct and arise in the interval between the first and second sounds. It is necessary to exclude heart bigeminism. Very rarely low humming sounds may develop at the base; their significance has already been discussed in connection with heart block.

2. *Arteries, Blood, and Blood Pressure.* The peripheral arteries are often stiff and tortuous. This is especially true of the temporals, which may be very rigid and prominent. Arterial degeneration is not invariably present, however, as a large number of patients appear to have vessel tissue of good quality.

So far as I am aware the blood characters have never been worked out. Hoffmann's patient had 55 per cent. of hæmoglobin. Lewy states that he was unable to count the cells, as every attempt to withdraw a specimen precipitated an attack.

Huchard thinks that the blood pressure is high during the attack. No definite statements occur in the literature.

3. *Urine.* Hoffmann states that the secretion of urine is diminished before an attack, but admits that the evidence is not conclusive. In a large majority of patients it is of rather low specific gravity, free from albumin and sugar, and relatively negative.

Course and Prognosis. The outlook will depend largely upon the age and causative lesion. When the complex develops after middle life as a functional disorder in the absence of organic heart disease, the prognosis is favorable as regards life and the condition may be well borne for years. The outlook is, as a rule,

favorable also in the abortive forms occurring in young individuals as the result of blood changes and psychical disturbances. Even in cases of more severe type the attacks may be withstood for ten years or more. Marked irregularity of the pulse, heart block, and other evidence of myocardial involvement are of the gravest import and may rapidly lead to a fatal issue. Death usually occurs in an attack.

TREATMENT. All attempts to cut short the characteristic attacks have so far been unsuccessful. Rest in bed should be enforced in all cases where the seizures are of daily occurrence. The foot of the bed should be elevated. Visitors, particularly those nearly related, should be excluded and every possible step taken to insure both mental and physical quiet. The diet should be simple and easily digestible. Tea, coffee, and tobacco must be absolutely prohibited.

Internal treatment is on the whole unsatisfactory. Potassium iodide in small doses—ten to twenty grains per day—is the most generally applicable remedy, and it may be given with benefit for long periods. Inhalation of oxygen is recommended by Hoffmann. Twenty-five to thirty litres may be taken daily for several months. The bromides are seldom of service. Digitalis is of doubtful value and must be used with extreme caution.

Residence in an equable climate is advantageous and should be recommended, together with instructions as to the avoidance of worry and physical exertion.

The pathological physiology of the Adams–Stokes complex is obscure. Of necessity it must remain a source of speculation until the causative factors are better understood. Taking into consideration the fact that a majority of all cases concern patients past middle life, and that it is customary to assume circumscribed or diffuse artery stiffness as an essential condition, it is natural to suppose that blood pressure studies would be instructive. On this important point, however, there are no authoritative statements in the literature.

The case I am about to present has been under observation nearly two years. During this period the object has been to accumulate trustworthy data on blood-pressure variations by making frequently repeated determinations. It will be necessary, therefore, to briefly describe the apparatus employed and the method of investigation, before proceeding with the clinical history of the patient.

All blood-pressure measurements were made with an improved form of the Riva-Rocci sphygmomanometer. This instrument is now so widely known that it will be unnecessary to describe it in detail. The apparatus is designed to register in millimetres of mercury the air pressure required to obliterate the pulse wave in the brachial artery. The arm band most commonly used in this country is about 5 cm. in width, made of thin rubber protected by a

jacket of canvas or duck, and is not provided with a rigid outer shell to prevent unequal distention. Müller (1902) in a simple theoretical discussion, made clear the inherent defect of this arm band and called attention to the fact that with it the normal values obtained are too high, ranging between 140 and 150 mm. He adopted in preference one designed by von Recklinghausen, which is less open to objection. This form consists essentially of a thin rubber cylinder 15 cm. in diameter, coated on the outer side with thin silk and applied around the upper arm by means of an ingenious double clamp. A strip of thin sheet brass of equal width is fitted smoothly over this and prevents unequal distention. With this band Müller obtained normal values varying from 105 to 115 mm. A similar band made for me by Zimmerman, of Leipzig, was used in all blood-pressure determinations carried out in the course of this investigation.

Observations were made, as a rule, with the patient in a sitting position. The band was first applied to the bared arm at the level of the heart, and the patient instructed to assume an unconstrained attitude. After from fifteen minutes to half an hour of rest, pressure observations were made in triplicate. Readings were taken under three sets of experimental conditions: 1. At once after walking ten blocks at a slight up grade, omitting the fifteen-minute period of rest. 2. After rest for at least a quarter of an hour in a comfortable position. 3. Immediately after taking a prescribed amount of active exercise—walking and running in a room three minutes with no period of rest. No difficulty was experienced in obtaining concordant readings, the maximum variation never exceeding 2 mm. Muscle movements of any sort introduce a gross error. For this reason it is impossible to determine blood pressure during typical convulsions.

Pulse tracings were taken with the ordinary Dudgeon sphygmograph. Unfortunately means were not at hand to determine the relation of the pulse curve to the respiratory cycle. A graphic presentation of the results, together with the conclusions deduced therefrom, will be introduced after reviewing the clinical history of the patient.

E. L., aged seventy-two years, a baker by occupation, applied for treatment at the Medical Dispensary of the University of California about June 5, 1902. He stated that the first symptoms of his trouble made their appearance in February of the same year. At that time he was working at his trade in a country town and was required to report for duty during the night. The actual onset was preceded for some days by indefinite symptoms. He was nervous, depressed, and had to urinate with unusual frequency during the day and four or five times at night. While on his way to work one evening he read a letter containing bad news; had what he describes as a rush of blood to the head, and fell unconscious to the ground.

The attack quickly passed off, however, with no unpleasant after-effects, and did not prevent him from engaging in his usual duties. Later in the evening, while stooping over the oven, an abortive attack was experienced, with a partial loss of consciousness and a feeling of suffocation and distress. Toward morning he again fell in a syncopal attack, but soon regained consciousness and was able to walk home. From this time on the seizures were of daily occurrence, forcing him within a short time to give up his work. A period of rest proved beneficial, but the trouble reappeared in May and induced him to consult a physician. The results of treatment were unsatisfactory. Indeed, under the influence of digitalis, which had been ordered for him, the attacks recurred with greater frequency, until finally, becoming alarmed about his condition, he moved to the city.

The following notes were taken at the medical clinic:

The patient was the youngest child in a family of nine. Both parents died at an advanced age. The father had been a hard drinker for years, but always enjoyed good health.

Patient was a delicate child. In his seventh year an ulcer developed on the posterior surface of the middle third of the right thigh, remained open several years, and from time to time discharged pieces of bone. Under treatment it gradually closed, but he was compelled to get about on crutches for several years. In his eighteenth year he had pleurisy, does not remember which side was affected, but declares that the condition recurred once a year for seven years. It finally disappeared after a severe illness suggestive of typhoid fever, which occurred in his twenty-fifth year. After this for a considerable period he enjoyed good health, except when under the influence of whiskey, which he has used to excess since his thirtieth year. At the age of forty-eight a painless swelling developed on the dorsum of the right hand. It proved of transitory nature, but has frequently recurred. There is no definite history of rheumatism, except that at times he has had some stiffness of the right hip and knee. In his sixtieth and again in his sixty-second year he had severe attacks of pneumonia. During the past nine years his health has been good, and he has worked steadily at his trade. He positively denies any venereal infection.

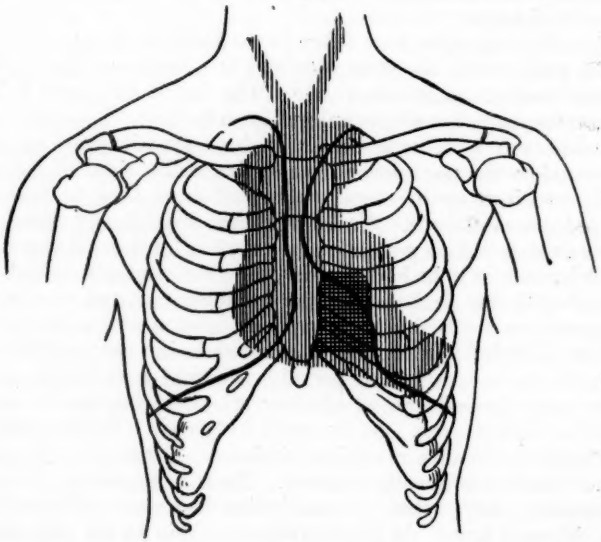
Examination. Short, slightly stooped man of moderate musculature, fairly well nourished. The skin is soft and of good color; a few purpuric spots are present on the upper arms and shoulders; there is slight dermographia. The facial expression is dull. The lower eyelids are baggy. The pupils are rather large, equal, and react briskly to light and accommodation. There is a well-marked arcus. The ears are negative. The tongue is clean, the palate well arched, and the throat free of abnormalities. The teeth are bad. The mucous membranes where visible are of good color.

The thorax is symmetrically developed and roomy, though stiff

and of limited mobility, the free costal borders slightly everted, the xiphoid angle wide. The apex impulse is neither visible nor palpable. On percussion the base of the heart is at the third cartilage, the apex half an inch inside the nipple line in the fifth interspace. The right border of dulness is at the left border of the sternum; the left border is 7½ cm. from the median line.

At the apex there is a musical systolic murmur, faintly heard also in the midaxilla, but inaudible at the point of the scapula. It is clearly heard at the junction of the eighth rib with the anterior axillary line. In the second right interspace there is a musical systolic murmur which can be clearly heard in the right carotid

FIG. 1.



artery, less clearly in the left. The tone qualities of this murmur exactly coincide with that heard at the apex. The most painstaking examination failed to reveal convincing evidence of multiple valve lesions. Starting from a point of maximum intensity midway between the apex and aortic region, the murmur can be traced over a wide area corresponding roughly to an outline including the heart and arch of the aorta, with a margin about three inches wide (see Fig. 1). The basic tones are clear, and there is no duplication. The second aortic sound is slightly accentuated. The tricuspid region is negative. There are no pulsations in the veins of the neck.

The radial and femoral arteries are neither hard nor tortuous, while the temporals, on the contrary, are rigid and prominent. The

pulse rate in the radial artery is 40, corresponding exactly to the apex beat. The pulse is occasionally intermittent.

The blood contains 10,000 leukocytes, 5,790,000 erythrocytes, and 88 per cent. of hæmoglobin. A second leukocyte count several days later gave 6600 cells.

The breath sounds are clear and very harsh, inspiration is somewhat prolonged, and there is slight dyspnœa on exertion.

The abdomen is flat, with no inequalities, and the reflexes are intact. There are no sensitive points. The liver is not enlarged; its lower border is smooth and barely palpable. The spleen is negative. The kidneys cannot be felt. The urine is clear, of light-amber color, sharply acid, and a normal mucous cloud separates on standing. The specific gravity is 1020. There is a faint trace of albumin, but no sugar. In the sediment there are large numbers of barrel-shaped crystals of uric acid. There is very little epithelium, not more than represents the normal desquamation from the bladder wall. A few unaltered red blood cells are present, but no pus. There are a few hyaline and finely granular casts.

The left thigh is smaller than the right and shows a linear, deeply depressed scar in the middle third posteriorly. In both legs below the knees there are a few small varicose veins, but no œdema.

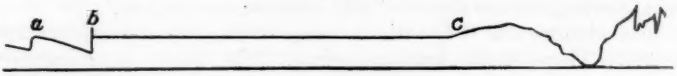
There are no disorders of sensation: heat, cold, and pain reactions are intact. The knee jerks are lively, the left more pronounced than the right. The cremasteric reflex is present, but very sluggish.

He is positive that his trouble has been greatly aggravated by the action of digitalis, which he has been taking for some weeks. When he first came under observation he had many attacks daily. It was not uncommon to count ten or more in an hour. At times the slightest disturbance—speaking harshly, or slamming a door—would precipitate a seizure. Raising the extended arm over the head was for a considerable time a certain provocative measure. He is able to predict the moment of onset by a sensation of blood rushing to the head, and at once adopts an attitude of absolute immobility. In the attack the head is held well down between the shoulders; the chin thrust forward; the eyes wide open, with dilated pupils; the expression very anxious. A chair or other support is suddenly grasped; consciousness is suspended; the respiration becomes stertorous; the body is bent forward on the hips; the arms and legs partly flexed and very rigid. This tonic condition is succeeded by irregular clonic contractions, which affect both extremities simultaneously, but quickly pass off, the whole attack lasting from five to fifteen seconds. The skin is moist with perspiration after the seizure.

The behavior of the pulse is characteristic. By careful palpation it is often possible to detect an oncoming attack. The pulse waves become less frequent, dropping from 40 to 26 or less, with perhaps slight arrhythmia, then fade out entirely, and the heart ceases to

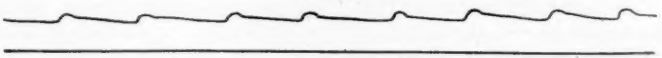
beat. By consulting Fig. 2 it will be seen that absolute heart rest occurs some time before the convulsive phase, since the slightest movement of the wrist tendons suffices to disturb the tracing device. At *c*, Fig. 2, the beginning of the muscle tremor is plainly indicated.

FIG. 2.



In order to obtain a tracing characteristic of the period of heart rest, it was necessary to apply the sphygmograph, await an attack, and then liberate the recording mechanism at the moment of heart rest. Unfortunately this gives no clue to the pulse characters before the onset. The sphygmograms taken in the free period vary very little in their general form. The tracing shown in Fig. 3 illustrates the

FIG. 3.



essential features, especially the gradual decline, with no dicrotism. The period of heart immobility does not terminate at the beginning of muscle movements; on the contrary, it persists throughout the attack. No sound could be heard over the præcordia at this time. Repeated attempts were made with the radiograph to investigate the heart movements during a seizure. Little of value was noted, however, owing to the suddenness of onset, brief duration, and the violent movements of the body.

A simple dietary, mostly liquid, was ordered, the tincture of digitalis withdrawn, and small doses of nitroglycerin substituted. After a week, no definite improvement being noted, the nitroglycerin was replaced by potassium iodide, five drops of the saturated solution three times daily. Within a short time marked improvement became apparent. The pulse, which previously had ranged between 30 and 40 in the free period, now rose to 72. The syncopal attacks became less frequent, and toward the end of July entirely ceased. The patient was dismissed from the clinic at his own request.

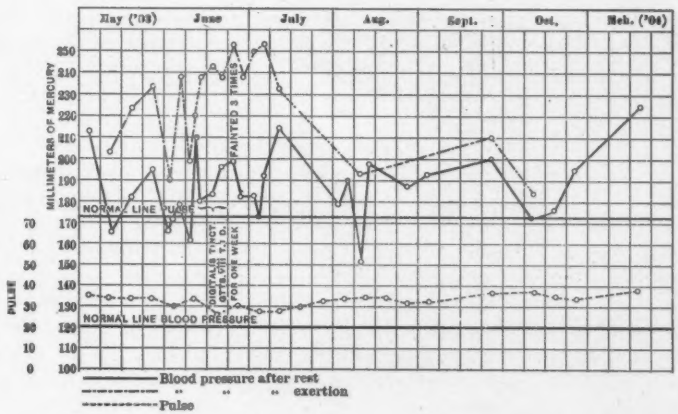
On October 11, 1902, he again applied for treatment, having had several dizzy spells during the past week. Reports that he stopped taking the iodide solution shortly after leaving the clinic. During September he had fair health, but latterly has been "seeing black." A physician who attended him recently ordered digitalis and nitroglycerin. Three days thereafter the typical seizures reappeared. He had two attacks while undergoing examination, which were rather less severe than those previously seen. At the wrist the

pulse rate was 62, quite regular, corresponding exactly to the apex beat. The heart condition was unchanged. The urine was intensely acid and contained large numbers of uric acid crystals. The amount in twenty-four hours was 1575 c.c., with a specific gravity of 1018. Between the 11th and the 23d of October he took twenty drops of saturated solution of potassium iodide every day. The pulse rate averaged 72; the subjective condition was good and he had no attacks.

On May 21, 1903, he reported that he had had no fainting spells since October. At times, especially in foggy weather, he has had some dizziness, but in other respects he has been quite well. There was no change in the heart condition. The pulse for some time had been very slow, rarely exceeding 40; on that date it was 36.

On March 7, 1904, the subjective status was excellent. He had been taking the iodide solution continuously since October, 1902. Occasionally, especially in foggy weather, he had spells of "seeing black." He stated that the condition sometimes persisted for a minute or more. There were no new objective signs. The heart was unchanged. The murmur had the same distribution and characteristics last reported. The pulse was 38 and regular. The skin was soft, clear, and of remarkably good color. The urine contained a trace of albumin, was intensely acid, contained large numbers of uric acid crystals, and its specific gravity was 1020. A few hyaline casts were found.

FIG. 4.



The blood-pressure observations were begun May 21, 1903, nearly a year after the patient was first seen, and some time after the last series of severe attacks. A graphic presentation of the results is given in Fig. 4.

The following general conclusions are drawn from the experimental data obtained:

1. The blood pressure is constantly very high, ranging from 170 to 250 mm.

2. The great arterial trunks of the extremities are fairly elastic.

3. Although very high blood pressure has existed for at least a year, the urine is normal in volume and specific gravity; there is little evidence of diffuse interstitial nephritis or of left ventricular hypertrophy. From this it follows that the high pressure is well borne by the heart and kidneys.

4. Active exercise, as a rule, has no accelerating action on the pulse. I have never counted an increase greater than two waves per minute. On the other hand it occasionally exaggerates the bradycardia.

5. Exercise invariably causes a sharp rise of pressure.

It seems difficult to harmonize such contradictory findings with the prevailing theory of the circulation. It will be sufficient to emphasize the most conspicuous anomaly, namely, the manifest disparity between the laggard work of the heart and the tremendous pressure at the periphery. This would appear to be too great to permit of explanation by a theory which assigns to the left ventricle almost the entire work of overcoming the inertia of the blood column. In the light of recent knowledge there is reason to believe that the arterial system and the vast capillary network of the periphery constitute a powerful secondary mechanism in constant rhythmic activity. It will be apparent that such a supposition denies that the heart wall generates the entire motive power, assuming on the contrary that other portions of the blood vascular system contribute a large quota of the driving energy. It is well to bear in mind in this connection that the whole arterial structure has perfect anatomical continuity.

Hasebroek (1903) has attempted to establish a new theory of the circulation, which gives this secondary apparatus a more definite value. Whether his dictum regarding the motor function of the peripheral vessels may be accepted is open to debate. There is confirmatory evidence in support of such a view, however, and it affords a logical explanation of a number of vascular phenomena that are otherwise almost wholly inexplicable. It would be entirely beyond the scope of this paper to consider in detail the arguments for and against such a view, but it is my belief that the so-called Adams-Stokes phenomena are highly suggestive of the existence of such an auxiliary mechanism dominated by the vasomotor system. The mode of action of psychic and other stimuli which are able to produce unstable equilibrium of this balanced heart and peripheral machine remains doubtful, but it is safe to assume that in the future this problem is destined to occupy a much more conspicuous place in the annals of research.

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BORATED FOOD AS A CAUSE OF LESIONS OF THE KIDNEYS.

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Of the various chemical preservatives of food, the two most commonly employed are borax and boric acid, and these are used very largely together. They are used so extensively that it is safe to say that not only in the cities and large towns where the commercial importance of chemical preservatives is so great, but in the country at large, persons of all ages are likely to receive frequent, if not daily, doses of one or of the other or of both. They are added to milk, butter, oleomargarine, and some forms of cheese; they are almost always present in opened clams and oysters; they are applied externally to fresh meats to prevent sliminess and to fresh and salted fish; they are important constituents of the brine in which meats are pickled and corned; their solutions are injected into hams and other pork products, and they are to be found in almost every known make of sausages in the market, even those which are put up in hermetically sealed cans. It is commonly and correctly supposed that canned foods, having been subjected to the process of sterilization, need no chemical preservative; but these agents are used in canned sausages, not to prevent decomposition, but because they increase the water-retaining capacity and prevent the sausage contents from contracting and thus causing the casing to appear loose and wrinkled.

As much as 0.87 per cent. of boric acid has been found by Popp and Fresenius[1] in canned sausage, and even 2.8 per cent. by Kister[2] in canned crab meat. American corned beef has been known to contain 3.87 per cent., and pork (see instance below) kept three weeks in borax has yielded 4.05 per cent., its innermost parts containing as much as 2.73 per cent. Polenske[3] found borax in every one of fifty-one specimens of American preserved meats, the majority of which contained more than 1 per cent. Thus, nineteen contained between 1 and 2 per cent.; thirteen from 2 to 3 per cent., and one as much as 3.36 per cent. Here again the boron compounds are used not wholly on account of their preservative influence, but because they do not withdraw water as salt does, and hence the meat tends less to become tough and stringy. Moreover, they preserve the natural color, while salt destroys it. It has been said, in answer to objections made by the German Government and others to the use of borax as an external application and as a constituent of pickling-brines, that that which adheres to the surface is easily washed away and that that which penetrates the tissues is removable by soaking. This, however, is not true. Once established in the tissues, it cannot be soaked out, nor even boiled out. Heinze[4] soaked for twelve hours a piece of meat containing 1.16 per cent. of boric acid, and found that even then it contained 0.93 per cent.; and Beythien and Hempel[5] washed a piece containing 1.12 per cent. for twelve hours in running water, and then recovered 0.69 per cent. Polenske[6] examined a 600-gram piece of fresh ham that had been kept three weeks in powdered borax, and found in the outer fat, 0.076 per cent.; in a piece cut from the middle, 2.73 per cent., and in a fair sample of the whole, 4.05 per cent. Two fresh hams, weighing twelve pounds each, were kept, one in boric acid and the other in borax, for four weeks; then they were washed, and from each was taken for chemical examination a slice 10 x 6 x 6 cm. It was found that into each one the chemical had penetrated very thoroughly, so that one may not say in a given instance whether the drug soaked in or was injected.

In milk the addition of from 0.1 to 0.2 per cent. of a mixture of the two is recommended, an amount which seems insignificant until we observe that it is equivalent to from 15 to 30 grains per quart. It is asserted that its presence in butter to the extent of 35 grains to the pound is necessary and harmless, and in several countries this admixture has received official sanction. With curious inconsistency, not one, but several, government commissions

[1] Zeitschrift f. Offentliche Chemie, 1897, p. 155.
[2] Zeitschrift f. Hygiene u. Infectionskrankheiten, 1901, Bd. xxxvii. p. 226.
[3] Arbeiten aus dem Kaiserlichen Gesundheitsamte, 1900, Bd. xvii. p. 561.
[4] Chemiker Zeitung, 1899.
[5] Zeitschrift f. Untersuchung der Nahrungs- u. Genussmittel, 1899, p. 842.
[6] Arbeiten aus dem Kaiserlichen Gesundheitsamte, Bd. 1902, xix. p. 167.

have decided that, although borax and boric acid are highly objectionable in milk, their presence in butter and concentrated milk to the extent of 35 grains per pound and in cream to the extent of 17.5 grains per pint is a commercial necessity of no sanitary significance. To clams and oysters opened at the place of taking and shipped in bulk to distant points, they are added with a generous hand. I have seen no reports of analyses, but I have found as much as 4.5 grams (reckoned as boric acid) in a pint of Norfolk oysters bought in Boston.

Concerning the preservative power of the boron compounds there is some dispute, but the fact remains that, for general purposes, they meet the requirements of trade better than any known substance. It is true that food already undergoing decomposition cannot be made sound, and that the process of putrefaction, once started, cannot be checked by either boric acid or borax; but it is equally true that, even though they do not kill bacteria, they inhibit their growth. Infected meat treated with these substances will keep much longer than if untreated, and, indeed, without losing its capacity for producing injury. Thus, von Fodor killed an animal twenty-four hours after it had been inoculated with anthrax, and treated a part of the meat with borax. After a number of days, the untreated portions became putrid, but the borated meat appeared to be normal in all particulars, although it was swarming with anthrax organisms.

When added to milk to the extent of 1 to 2 grams per litre (0.1 to 0.2 per cent.), it has no very great influence in postponing souring, although with proper precautions, in themselves influential, coagulation may be deferred as long as three days; but under ordinary conditions the accomplishment of this result would require the presence of so much of the preservative as to destroy the natural taste.

Whether or not the boron compounds are capable of causing injury to the consumer of borated foods, has been the subject of much experimentation and heated controversy. Reviewing the literature, one is struck at once by the fact that adverse opinions are based largely upon occasional untoward results of boron medication, upon feeding experiments extending over but a few days, and upon *a priori* reasoning, and that favorable reports are based upon equally insecure foundations. A very large part of the experimental work has been limited to the determination of their action upon digestion and metabolism, and most of the researches have been of very short duration. Many of those interested on both sides of the question have been content to make a few observations, and then have dismissed the subject with a wave of the hand as settled for good.

It is not to be supposed that any food preservative that will produce obvious injury within a few days or weeks can ever come into

general use; and an experiment lasting but a few days and involving the introduction into the system of but a few grams of a chemical not classifiable as a poison can have no probative force. For example, an experiment much quoted by the opponents of boron and fiercely assailed by its friends is that of Kister,[1] who gave 3 grams of boric acid daily to three men, one of whom was made sick at once with vomiting and diarrhœa, and withdrew; a second had albuminuria on the fourth day, when the administration of the drug was discontinued and the albuminuria disappeared; and the third lost his appetite on the seventh day, had albuminuria on the tenth, ceased to take the drug on the twelfth, and became normal on the thirteenth.

Neither of the subjects whose service lasted beyond the first day was given more of the drug to ascertain if a causal relation existed between it and the albuminuria, which may have been an incident of any one of a number of conditions. Another, ascertaining that a food of somewhat complex composition (blanc mange) was made partly with milk which contained borax, attributed the sickness of a number of persons who ate of it, and the death of some fowls that consumed the remainder, to the toxic nature of borax, without considering other possibilities or trying the effect of borax alone on other fowls. Others, reviewing the literature, have brought together the various effects that have been observed in subjects of experiments and in patients with idiosyncratic tendencies, and have proceeded to draw unjustifiable conclusions. Thus, one insists that the results of eating borated foods are vomiting, diarrhœa, diuresis, lesions of the skin, interference with absorption of food, excretion of abnormally large amounts of nitrogen, and loss of weight.

It is well known that certain individuals cannot undergo borax or boric acid medication any better than certain others can take quinine or morphine, or eat lobster and strawberries; and when we read, as we may in recent medical literature, of ill-effects from the use of a 2 per cent. solution of boric acid as a mouth-wash, of skin eruptions following repeated washing of the bladder with boric solution; of nausea, loss of appetite, and depression following the dressing of open wounds with boric ointment; of total baldness following internal administration of 10 to 20 grains daily for five weeks, and of death from washing out a stomach with a 2.5 per cent. solution, we recognize that these must be isolated cases of idiosyncratic susceptibility from which it would be highly unscientific to generalize. It is often charged that borax and boric acid cause diarrhœa, but such a statement deserves but little consideration. It may occasionally happen in experiments that the subject is purged, but it must be remembered that in the present

[1] Zeitschrift f. Hygiene u. Infectionskrankheiten, Bd. xxxvii. p. 225.

state of the food supply one is likely any day to swallow more or less of both with fish, oysters, ham, corned beef, sausage, milk, and butter, and yet we are not continually purged. Indeed, those who are chronically constipated doubtless absorb borates frequently without relief.

On the other hand, experiments of short duration yielding no evidence of injury to digestion have been accepted as demonstrating the absolute innocuousness of the boron compounds as food preservatives, regardless of the fact that they prove little or nothing as to possible effects of long-continued ingestion on the different organs of the body. It is true that, so far as they go, the weight of evidence of digestion and metabolism experiments is slightly in favor of the defendant substances; but such experiments, as a rule, are not carried far enough. Concerning the value of short researches of this character Liebreich,[1] who is doubtless the most strenuous advocate of the use of the boron compounds in foods, says: "It must always be emphasized that no absolute conclusions are admissible after a single series of experiments of such short duration, particularly with experiments on metabolism, which in themselves call for strict criticism, especially in the case of human beings."

As has been stated above, no substance capable of inducing acute symptoms of any kind could long be employed as a food preservative. The use of a substance, for example, which would cause nausea and vomiting, or diarrhœa, or headache, or eruptions of the skin, or other symptoms, which, occurring in large numbers of people at the same time, would direct attention to the food supply, would necessarily be of short duration. With such as cause no such effects, it is manifestly absurd to institute short researches with any expectation of positive results. The preservatives in common use are either harmless or they produce injury insidiously on long-continued ingestion; and in the latter case it is not to be expected that every individual subjected to their influence will be affected to the same extent.

Owing to the fact that one cannot possibly have a record of the nature and amounts of the various substances that for weeks and months and years have been taken into the system, and that the influence of this and that condition to which one has been subjected cannot be measured, it is not possible in case of discovery of the existence of pathological processes definitely to trace the cause thereof back to food. Let us suppose, for example, that A. B., after some weeks of not feeling well, consults his physician and undergoes a thorough examination, in the course of which are found albuminuria and other evidence of degeneration of the kidneys. Is it likely that his condition will be attributed to the character of

[1] Second Treatise on the Effects of Borax and Boric Acid on the Human System, London, J. & A. Churchill, 1902.

his food? And even if it were, would it be possible, in the absence of samples of everything that he has eaten for a long time, to single out the particular components of his diet which, nowadays, is likely to contain so many representatives of the pharmacopœia? Manifestly not.

In all that has been published concerning the influence of borax on the system, the most striking facts have come not from metabolism experiments, but from its therapeutic use. It has repeatedly been shown, notably by Dr. Féré, that the medicinal use of borax is not unattended with risk of producing or aggravating lesions of the kidneys. Liebreich,[1] however, stated in his first communication concerning the effects of boron compounds on the human system that weak and diseased kidneys bear borax and boric acid particularly well. In his "Second Treatise," he relates that to each of eight rabbits a single intravenous dose of borax was given without producing any evidence of renal irritation, and he says, therefore, in heavy type: "From these experiments on animals it may be deduced that borax is entirely innocuous as regards the functions of the kidneys." He gave boric acid to seven patients in varying amounts and for different periods. Two received 28 grams in seven days; three received 33 grams in eleven days; one took 37.5 grams in twenty-two days, and another 39.9 grams in twenty-four days. Daily examination of the urine revealed no albumin. To five others he gave borax in smaller doses (14.1 grams in twenty-six days, 38.7 in thirty-two days, 60.9 in thirty-seven days, 24 in fourteen days, and 23 in fourteen days). In two of these patients, slight temporary albuminuria was observed. To another man he gave 3 grams of borax on each of three days about two weeks apart (November 8th and 22d and December 2d). Examination of the urine on November 19th and 27th and December 8th showed no albumin. From the results observed in these three brief experiments he says, quite unmindful of his above-quoted statement concerning absolute conclusions from single series of experiments: "The above-named investigations offer a further proof that borax and boric acid are substances which cause no injury to health when judiciously used." Without further discussion of the clinical evidence of the possibility that the boron compounds may injuriously affect the kidneys, I will submit the details and results of a feeding experiment which I have lately conducted for a period of nineteen weeks.

Twelve healthy male cats were selected and kept under precisely the same conditions in separate cages. All were fed on the same food with this difference: One (No. 1) received no preservatives; six (Nos. 2, 3, 4, 5, 6, and 7) received borax to the extent stated below, and the remaining five (Nos. 8, 9, 10, 11, and 12) received

[1] Vierteljahrsschrift f. gerichtliche Medicin, 1900, p. 83.

another preservative which proved to be innocuous. The cats that were fed on borated food received the following amounts of borax:

No. 2	. . . .	72.5 grams	in	133	days.	Average dose		0.544
" 3	. . . .	112.0	"	133	"	"	"	0.842
" 4	. . . .	28.0	"	42	"	"	"	0.667
" 5	. . . .	108.0	"	133	"	"	"	0.812
" 6	. . . .	106.5	"	133	"	"	"	0.800
" 7	. . . .	48.0	"	56	"	"	"	0.857

Of the dozen animals under observation, but three showed any sign of illness, and they were of the borax group. No. 4 sickened very soon, and died at the end of the sixth week; the other two were sick occasionally, but during the last weeks of the experiment they appeared to be normally active. Each subject was weighed at the end of each week; all showed occasional losses, but with the exception of No. 4 there was a net gain ranging from 230 to 990 grams.

At the expiration of the periods above mentioned, all of the animals were killed (No. 4 died as stated), and in each case the heart, lungs, liver, spleen, kidneys, lymph nodes, bone-marrow, pancreas, thyroid, thymus, adrenal, large bloodvessels, striated muscle, stomach, and small intestine were examined microscopically by Dr. E. E. Tyzzer, of the Department of Pathology.

The control cat (No. 1) showed no lesions whatever. Cat No. 2 showed a slight cirrhosis of the liver and nothing more.

Cat No. 3 showed kidney lesions, described below, and a slight increase of fat at the periphery of the lobules of the liver.

Cat No. 4 showed kidney lesions, described below, a small contracted spleen, slight increase in connective tissue in the liver, multiple adenoma of the pancreas. The lymph node showed increased fibrous tissue and was filled with epithelioid cells.

Cat No. 5 showed kidney lesions, described below, and fatty, but not markedly, degenerated liver.

Cats Nos. 6 and 7 showed kidney lesions, described below, and minute foci of lymphoid cells in the liver, generally peripheral to the lobule.

Cats Nos. 8 to 12 inclusive, which were given another preservative, showed no marked changes in the kidneys and no important or constant lesions elsewhere. Except that so far as the kidneys are concerned they may be regarded in the light of additional controls, they have no place in the present paper.

Thus, of the twelve cats, only five showed any marked changes in the kidneys, and these were all members of the borax group, only one (No. 2) of which escaped. The fact that five of this group, and none other, showed marked changes in the kidneys is significant; it cannot be looked upon as a matter of mere coincidence.

The stained sections of the five kidneys were submitted to Professor William T. Councilman, who very kindly reported thereon as follows:

"Five kidneys. All show lesions of the same general character, but differing in intensity. They consist, first, in degeneration of epithelium in all tubules, but most marked in the convoluted tubules. The normal fat vacuolation is very greatly increased, and in addition there is swelling and, in places, disintegration of epithelial cells, with fragmentation of nuclei. In the lumen of many of the collecting tubules are round and irregular granular masses representing cell fragments coming from higher up. Tubules also are found almost completely stripped of epithelium. In one kidney the change is entirely confined to degeneration of intense character, with hyaline casts in the small tubules. In the other cases the degeneration is not so intense, but is accompanied by foci of cellular infiltration. The largest of these foci is 2.5 by 3 mm. The infiltration is chiefly marked at the cortex and also around single glomeruli. Where this

FIG. 1.

Foci of intense interstitial infiltration at the cortex, with degeneration and destruction of tubules. (15 diameters.)

is most intense, the tubules are entirely destroyed. In one large area in the pyramid there is circumscribed necrosis with invasion of phagocytic cells. These lesions are analogous to those found in subacute and chronic nephritis in man, although they do not conform to any definite type."

It may be objected that the daily doses of borax given were very large and, in proportion to weight, much greater than a person could possibly receive in food in the course of a day, but that is not so, and, moreover, it must be remembered that this substance is asserted to be absolutely innocuous even in large doses, and, indeed, rather beneficial than otherwise. Thus, it is said that for years Virchow took a daily dose of a gram, and

in the last years of his life 2 grams with benefit, though in what manner is not stated. But one can easily swallow, without know-

FIG. 2.

Focus of cellular infiltration in pyramids, with complete necrosis of tubules and invasion of phagocytic cells. (15 diameters.)

FIG. 3.

Focus of cellular infiltration around glomerulus, with degeneration and hemorrhage in adjacent tubules. (100 diameters.)

ing it, very much larger amounts in a day, and even with a single meal. We have, for example, the following known possi-

bilities: milk, 2 grams per quart; butter, 2 grams per pound; cream, 1 gram per pint; oysters, 4.5 grams per pint; corned beef, 18 grams per pound; ham, 18 grams per pound; sausages, 4 grams per pound. An oyster stew made with a pint of such milk and a quarter of a pint of such oysters, followed by a quarter of a pound of such ham or corned beef, would constitute a not extraordinarily hearty meal for an average man, who would ingest therewith upward of 7 grams of borax and boric acid, an amount which in the course of a day's food might easily be doubled.

INHALATION OF GASOLENE VAPORS.

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Some years ago (1897) I began an investigation of the effects of gasolene vapors, with a view to finding a cheap anæsthetic for laboratory work. The results showed that this drug would not be practical, and I have delayed their publication on this account. The renewed interest in the subject, aroused by the employment of petroleum ether in Schleich's inhalation mixture, causes me to submit a brief summary of my work. I employed the commercial gasolene in most of the experiments; in a few I used a purer sample of petroleum ether. The effects were identical. These coal-oil products consist of a mixture of various hydrocarbons of the series CnH_{2n+2} and CnH_2n, the principal ingredient being pentan (C_5H_{12}). The experiments were confined to dogs and frogs.

Effect on Normal Dogs. When the gasolene is poured on cotton, applied to the mouth of a dog, and covered with a towel, the very first effect is a deepening of the respiration. The animal then struggles violently to escape, but the movements soon lose their purposive type, and degenerate (within two to ten minutes) into inco-ordinated convulsions. The animal has apparently lost consciousness, and may be laid on the table. The four legs paw the air as in running. The eyelids, the tail, and the toes twitch. The pupils dilate widely. These rather weak convulsions then assume a stronger and more general type, clonic spasms alternating with tetanus. The neck muscles develop a surprising strength. During this convulsive stage the animal appears quite insensitive to pain, but the corneal reflex is not quite abolished. In the intervals between the general convulsions the animal lies flat, apparently exhausted, with constant twitching of individual muscles.

Death occurs suddenly in two to twenty minutes, heart and respiration stopping at nearly the same time.

The general course of the intoxication was very uniformly, as described, in four normal dogs. In a fifth animal, however, in which death occurred in seven minutes, the convulsions were more of the ordinary asphyxial type.

The autopsy of the animals killed by the inhalation of gasolene gave also very uniform results. The veins and the heart, especially the right side, are considerably distended. The lungs and skeletal muscles are conspicuously pale; the kidneys are injected. The liver, spleen, and intestines appear normal. The blood remains fluid for some time after death. It does not show any striking microscopic or spectroscopic changes.

The previous injection of a heavy dose of morphine does not modify the phenomena if the gasolene is pushed (one experiment); but it makes it possible to graduate the effects and to extend the inhalation over some time.

An experiment made in this manner showed that even a dilution of 1:5 causes some anæsthesia and some convulsions. The anæsthesia was not, however, very deep. Recovery followed promptly on discontinuing the administration. The protocol of this experiment follows:

Experiment. At 10.59 began the administration of 1 volume petroleum ether to 5 volumes of alcohol.

In three minutes there are convulsions, pupils large, some anæsthesia, cornea sensitive.

Discontinued gasolene and started artificial respiration.

In one minute the animal raises its head, and in two minutes it walks around.

At 11.06 administered petroleum ether 1 to alcohol 1.

In twenty-eight minutes twitching of eyelids and lips, tongue blue, general convulsions, pupils rather large, very little anæsthesia.

Artificial respiration begun. The pupils become normal long before the twitchings disappear. In six minutes the animal is still weakly anæsthetic; in eleven minutes it runs about, but is very narcotic.

Effect of the Inhalation of Gasolene on the Circulation and Respiration. For this purpose the animals were first put under a preliminary anæsthesia by a heavy dose of morphine and sufficient A. C. E. mixture. A carotid artery was connected with a kymograph. The trachea was supplied with a cannula, and this was connected with a T-piece, one end of which led to a Woulfe's bottle containing the anæsthetic (or gasolene); the other end leading to a Marey's tambour which registered the respiratory movements. The experiments were made on four dogs.

Mixture of Gasolene 1 *to Alcohol* 8 (*volumes*). The animal remained perfectly anæsthetized for two hours without noticeable

change in the blood pressure. At this time the characteristic convulsions set in. The A. C. E. mixture was substituted, and the convulsions disappeared.

Gasolene 1 *to Alcohol* 5. No effect in fifteen minutes beyond a slight slowing of the heart.

Gasolene 1 *to Alcohol* 3. Immediate weakening of the heart, with fall of the mean blood pressure. Respiration greatly deepened and somewhat slowed.

Undiluted Gasolene. Three experiments showed an immediate weakening and quickening of the heart, with corresponding fall of blood pressure. Section of the vagi in the one experiment in which it was tried did not cause further quickening. The respirations were increased in depth and frequency. One animal died suddenly in ten minutes; the second died in thirty minutes, death being preceded by a sudden fall of blood pressure. The third animal survived over one hour, when it was killed by asphyxia. The last two animals showed a number of the typical convulsions, occurring first respectively three and seven minutes after beginning the administration. During the spasm the heart force increased somewhat, and the blood pressure rose greatly. In the intervals between the convulsions the blood pressure showed a further fall, and muscular twitchings persisted.

It is seen that dilutions of 1:5 are practically devoid of any effect on the circulation. The convulsions occur late. Some degree of anæsthesia can be maintained. With greater concentrations the phenomena consist in an increase of respiration (especially depth) and in a fall of blood pressure. The latter is at least partly attributable to a weakening of the heart. The tonic vagus inhibition is depressed or abolished. The convulsions cause a considerable vasomotor stimulation. Death occurs through vasomotor paralysis (with respiratory failure?), but is very late in some cases.

Effect of the Anæsthetics on the Convulsions. The preceding protocols show that the convulsions can occur after A. C. E. anæsthesia. I have, however, three records in which the convulsions were absent. In one of these asphyxia also did not produce convulsions. It may therefore be assumed that the absence of the convulsions in these cases was due to the persistence of the A. C. E. anæsthesia, which, as we know, lessens the effects of many convulsant poisons.

Effect on Frogs. Three experiments were made. The animals were placed under a bell-jar, the air of which was kept saturated with the vapor by a wad of cotton impregnated with the fluid.

Commercial gasolene was used in the first two experiments. The effects were uniform. The animals at once became excited, tried to escape, wiped the nose with the forepaw, etc. Soon paralytic phenomena were noticed, and in two and one-half to four minutes the animals could not turn when placed on the back, although they made ineffectual attempts. When put in water they could not main-

tain their position, floating with head upward. They would make an occasional fairly powerful stroke, but turn on their side in doing so. Replaced in the jar they would sit flat and execute occasional swallowing movements. In ten to fifteen minutes they would lie flaccid, with legs extended, but react weakly to stimulation. This excitability was also gradually lost, and the animals appeared dead, but on exposing the heart it was still beating strongly. The third frog was treated with petroleum ether, and the same phenomena developed, but more slowly. None of the animals exhibited any convulsions.

Results of Other Investigators. The older literature of gasolene inhalation is summarized by Joseph.[1] The first two cases were of laborers poisoned in cleaning gasolene tanks. The effects were doubtless in large part asphyxial. Some animal experiments of Felix are quoted. Some experiments were also made on criminals, in whom complete anæsthesia was produced.

Elfstrandt[2] investigated pure pentan. The effects on frogs, rabbits, and cats agree with those reported by me. Death occurred by paralysis of respiration, and complete recovery resulted on artificial respiration. The anæsthetic action is comparatively weak. The blood pressure in rabbits is lowered to a greater extent than by ether anæsthetic.

Meltzer[3] notes the convulsions, but saw no anæsthetic effect. With the latter conclusion I must disagree.

Kochmann[4] ascribes to petroleum ether a very weak anæsthetic action.

Conclusions. Gasolene (or petroleum ether) when inhaled has a comparatively slight anæsthetic action. The anæsthesia is, however, very dangerous, since the substance also produces convulsions, weakening of the heart, vasomotor depression, and paralysis of respiration. The kidneys are also irritated. The effects are produced very promptly, and recovery under artificial respiration is also prompt.

The cause of the convulsions is not known. They do not occur in frogs.

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[2] M. Elfstrandt. Arch. f. exp. Path. and Pharm., 1900, Bd. xliii. p. 435.

[3] S. J. Meltzer, 1898. Cited by W. Meyer, Journal of the American Medical Association, 1903, vol. xl. p. 555.

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A CASE OF SUTURE OF THE HEART, WITH RECOVERY.*

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I am indebted to Professor Keen for the privilege of operating upon and reporting the following case:

T. E., aged twenty years, colored, cook, was admitted to the Jefferson Hospital, February 29, 1904. He had been stabbed in the chest with a long, rusty pen-knife. The stab caused severe pain, but neither felled him to the ground nor caused him to feel faint. He walked without assistance to the hospital, a distance of one and a half squares, then began to feel weak, and was found crawling up the steps leading to the entrance of the hospital. After being disrobed a small wound was found just above the third rib, about one inch to the left of the left margin of the sternum. The wound was surrounded by an emphysematous swelling, and bled continuously, the stream of blood being accelerated by each expiration. The temperature was 97°, and the pulse 80, empty and markedly irregular in volume and rhythm. He lay on the right side, breathing forty times per minute in a short, jerky manner. At times he would complain of a little pain and severe dyspnœa. He was sweating and very pale, and had vomited once. There was neither cough nor blood-spitting. Entire left chest was tympanitic on percussion, except for a small area around the wound. On auscultation the heart beats could be heard indistinctly; there was no bruit nor splashing sound. Under ether anæsthesia operation was begun about forty-five minutes after the infliction of the injury. Time of operation about forty-five minutes. Amount of ether, four ounces. An incision was carried along the second rib for four inches to the sternum, then down the left margin of the sternum to the fourth rib and outward along the fourth rib for four inches. The musculocutaneous flap was dissected back and the opening in the chest found between the third and fourth ribs, the knife having descended after passing through the skin. The exposure was then made more complete by continuing the sternal incision downward for two inches, and the triangular flap thus formed retracted. The third and fourth ribs were severed and forcibly turned toward the right, fracturing the costal cartilages near the sternum. The knife had passed through the pleural cavity and wounded the anterior edge of the left lung. The lung had collapsed into the vertebral gutter, and the pleural cavity contained a large quantity of clotted and

* Read before the College of Physicians of Philadelphia, May 4, 1904.

fluid blood. The opening into the pericardium was easily found by the spurting of blood with each pulsation of the heart; it measured about three-eighths of an inch. The pericardial wound was enlarged in the axis of the heart, and a penetrating wound of the anterior wall of the left ventricle found. This wound was parallel to the axis of the heart, nearer the auricle than the apex, and measured about three-quarters of an inch (larger than either the skin, intercostal, pleural, or pericardial wounds). Bleeding from this wound was free and continuous, whether more marked during diastole I am unable to state, as the heart was now beating very rapidly and resembled a quivering mass of muscle. The wound was closed with a continuous silk suture (six stitches), the sutures being inserted and tied without reference to diastole for the reason already stated. During the suturing the coronary artery was punctured by the needle, causing a profuse hemorrhage which was controlled by an additional suture. The pericardial and pleural cavities were cleared of blood, and the pleural cavity was irrigated with salt solution. The pericardial wound was sutured with a continuous silk suture, a small opening being left at the lower end for the passage of a gauze drain. No attempt was made to suture the lung (which was not bleeding) nor the parietal pleura. A gauze drain was also placed in the pleural cavity, gaining exit at the lower part of the primary incision. The severed ribs were approximated and the soft parts sutured with silkworm-gut. During the operation twenty-four ounces of salt solution containing adrenalin were injected into the circulation and strychnine and atropine were administered subcutaneously. After the operation the foot of the bed was raised, external heat applied, the extremities bandaged, and owing to the violent struggling of the patient morphine was given. Temperature, 100.4°; pulse, 150; respiration, 32.

Second day: Temperature, 103.4°; pulse, 130; respiration, 56. Dressing saturated with blood-stained fluid. Unproductive cough. Strychnine, whiskey, codeine. Delirium, subsultus, carphologia. Great pain in left chest, garrulous wound.

Third day: Temperature, 100°; pulse, 130; respiration, 46. Drains removed and replaced between the edges of the skin wound because of oozing of blood.

Fourth day: Temperature, 102°; pulse, 140; respiration, 52. Passage of air through the external wound has ceased. Ammonium carbonate and digitalis.

Fifth day: Temperature, 102°; pulse, 130; respiration, 60. Examination of the chest reveals dulness, rough breathing, and crepitant rales over the lower part. Blood: erythrocytes, 3,630,000; leukocytes, 38,800; hæmoglobin, 58 per cent. The chest was explored with a needle, with a negative result.

Sixth day: Temperature, 102.8°; pulse, 140; respiration, 48. Pus under skin flap; removal of several stitches.

Seventh day: Temperature, 103.6°; pulse, 140; respiration, 52. Cough continues; little pain.

Eighth day: Temperature, 103°; pulse, 150; respiration, 50. More profuse discharge of pus from the wound.

Ninth day: Temperature, 103°; pulse, 136; respiration, 48. Chest again explored for pus, with negative result.

Tenth day: Temperature, 103°; pulse, 128; respiration, 52. Prolonged paroxysm of coughing.

Eleventh day: Temperature, 103.4°; pulse, 130; respiration, 50. Remaining stitches removed.

Twelfth day: Temperature, 103°; pulse, 128; respiration, 50.

Thirteenth day: Temperature, 100.8°; pulse, 120; respiration, 40. Small slough separating from the skin-flap.

Fourteenth day: Temperature, 100°; pulse, 120; respiration, 40.

Fifteenth day: Temperature, 101°; pulse, 120; respiration, 28. Cough almost gone; never any expectoration. Thereafter the temperature, pulse, and respiration gradually fell until on the twenty-second day the temperature registered 99°, the pulse 98, and the respiration 20. Subsequently the temperature, pulse, and respiration remained below these points. On the thirty-fifth day the patient was allowed out of bed. On the fifty-sixth day the patient was discharged to return to the Out-patient Department for dressing, a small sinus leading down to the point where the ribs were severed, still persisting; this sinus closed a few days later. The ribs have united firmly, with little callous formation. The pulse varies between 80 and 90. Adventitious sounds have never been heard in the heart. The left chest expands very little on inspiration, and slightly roughened breathing, but no rales, can still be heard.

In olden times a wound of the heart was considered synonymous with inevitable and immediate death. Ellsberg[1] tells us that prior to the sixteenth century Hippocrates, Celsus, Paul of Ægina, Roland, and Lanfranc all mention heart wounds, and agree that they are invariably fatal. In the early part of the sixteenth century Fallopius[2] studied the subject, with a view to recognizing the cavity injured, and Jacob Hollerius made the assertion that a wound of the heart was not necessarily fatal. Later in the century bullets, arrowheads, and other foreign bodies were found in the hearts of animals which had continued to run for some time after receiving their death wounds, and tales were told of men who during the intoxication of battle continued to fight after being stabbed through the heart. Toward the end of the century Ambroise Paré[3] related the oft-quoted case of the Turin gentleman who received a rapier thrust below the left nipple during a duel, and who made several passes and pursued his slayer 200 paces before falling to the ground a corpse.

In 1642 J. Wolf[4] gave the first reliable account of the healing of a wound of the heart. Morgagni[5] (1761) stated that the cause of death in many cases was compression of the heart by the effused

blood, a theory which has since been converted into a fact mainly by the observations of Rose and the experiments of Cohnheim. It is interesting in this connection to recall the fact that Morgagni died of rupture of the heart.

Sherman[4] says Desault laid down the rules for pericardotomy for empyema in 1798, and it took surgery ninety-eight years to pass from the pericardium to the epicardium across a space that is such only potentially.

Benjamin Bell[2] (1783) did not believe healing could occur. A. Richter[3] (1786) considered the probe dangerous, and insisted upon absolute rest and free bloodletting. Larrey and Dupuytren laid down many rules for treatment. During the seventeenth and eighteenth centuries papers would appear describing cases in which protracted periods intervened between the injury and dissolution.

In the beginning of the nineteenth century Jamain, Zannetti, and others wrote on the viability of patients after heart wounds; but it was not until 1867, when George Fischer[5] published his classical study of 452 human cardiac wounds, that a firm foundation was laid for the evolution of the surgery of the heart. Fischer clearly established the fact that an individual might exist for hours or even days with a wounded heart; and that, indeed, in from 7 to 10 per cent. of the cases the wound heals and the patient actually recovers. Loison and Ollivier collected additional cases and corroborated the findings of Fischer. Ninni[6] states that according to the statistics of Jamain, Fischer, Lantenelet, and Zannetti, death is instantaneous in 18 per cent. of the cases.

Up until the closing years of the nineteenth century the treatment of wounds of the heart consisted in the application of an occlusive dressing, absolute rest, ice or blisters to the præcordium, sedatives, such as veratrum viride, digitalis, opium, and venesection. Rose[7] advised paracentesis of the pericardium or pericardotomy to relieve compression of the heart the result of a wound. In 1881 John B. Roberts suggested the propriety of attempting to suture a wound of the heart. Later Koenig and Delorme voiced the same opinion. The idea, however, was generally regarded as the wildest fancy of ultra-enthusiasts. Billroth declared that no surgeon who wished to preserve the respect of his colleagues would ever attempt to suture a wound of the heart. Riedinger (1884) considered the proposition below the dignity of mention. Tillmanns was convinced of the hopelessness of surgery when confronted with a bleeding heart. That the heart could be exposed and manipulated has been demonstrated by many experimenters. In 1894 Kronecker and Schmey[1] discovered that this organ might be punctured with but little interference with its action, except when a spot in the interventricular septum was touched, when immediate death ensued (Kronecker's co-ordination centre). In 1895 Rosenthal[1] essayed to treat an experimental wound of the heart by direct means—he used

a tampon of iodoform gauze, and the dog survived. During the same year Del Vecchio[1] was the first to suture the canine heart with success. In 1896 Salomoni[1] and in 1897 Bode[1] successfully closed wounds of animal hearts by suture.

Since then there have been many experimental investigations into this subject. The conclusions of Elsberg,[1] which may be taken as representative of the findings of the experimenters, are as follows: That the heart could be grasped with the hands or forceps and gently compressed, with no appreciable difference in its action, and that it could be penetrated by a needle and knife, producing only a temporary irregularity of its action; that penetrating wounds produced during systole bled more than those produced during diastole, and that wounds of the ventricles produced during systole were larger than those produced during diastole; that oblique wounds bled less than perpendicular wounds; that wounds of the right ventricle were more dangerous than those of the left because the wall of the right ventricle was thinner, and also because the blood in the right side of the heart coagulated more slowly; that wounds of the heart heal kindly and that the cicatrix is usually complete in about fourteen days; that interrupted sutures are better than continuous ones; that the tissue included in the suture always atrophied, and was replaced by scar; a smaller number of muscular fibres was included in the interrupted than in the continuous suture, thus producing less atrophy and less scar tissue; that superficial sutures were less likely to tear out than deep ones, and that the sutures should be inserted and tied during diastole because of the danger of their tearing out if tied during systole.

After the road had been paved by the knowledge that patients would often live long enough for operative interference, and that the heart would tolerate manipulation, two unsuccessful attempts were made in 1896 to close human cardiac wounds by suture, one by Farina and one by Cappelen, and in 1897 Rehn published the first successful cardiorrhaphy in man. Including these cases and the one reported in this paper, I have been able to find 60 cases with the surprisingly high recovery rate of 38⅓ per cent.

Wounds of the heart may be produced by penetration from without, as gunshot or stab wounds, or by fractured ribs, and by foreign bodies from the œsophagus, stomach, or bronchus. The heart may burst as the result of blunt force to the thorax or epigastrium with or without laceration of the pericardium, and it may rupture spontaneously (disease of myocardium or coronary artery, neoplasms, gummata, echinococci, abscess, aneurysm, etc.). That an attempt should be made to save as many of these cases as survive for a sufficiently long period of time after the accident needs no argument. Of the cases thus far recorded, 55 were stab wounds and 5 gunshot wounds. Two of the gunshot wounds recovered (Launay, Noll) and in two of the fatal cases the posterior wound was not

sutured (Bufnoir, Stern). As late as 1903, however, Wolff[8] advises expectant treatment in gunshot wounds of the heart.

The symptoms are those of acute anæmia or of compression of the heart, depending upon the escape of blood into the pleural cavity or externally or upon its retention in the pericardium. In some cases the patient may walk or even run for a considerable distance before falling to the ground. In a recent stab case in Philadelphia the patient was examined by a careful and competent surgeon, who regarded the wound in the chest as trivial because of the absence of symptoms. The patient died within twenty-four hours and an autopsy revealed a punctured heart. On the other hand, a patient may present marked symptoms without a serious injury. During the past year I was called to the Pennsylvania Hospital to see a boy who had been stabbed in the cardiac region. He presented all the classical symptoms of severe shock. Under a local anæsthetic the wound was enlarged and found to be non-penetrating. His symptoms disappeared in a few hours. In any case there may be nervous symptoms, such as vertigo, syncope, convulsions, vomiting, or hemiplegia (Pirogoff). The nervous phenomena are said to be more common in injuries to the left ventricle, and to be due to anæmia of the spinal cord. Respiratory difficulties are more marked in wounds of the right ventricle, but may be due to nervous influences, pneumothorax, or compression of the heart. Hæmoptysis is rare, even if the lung is injured.

When the blood escapes into the pleural cavity (the pleura was injured in 57 of the 60 cases, 95 per cent.) there will be in addition to the symptoms of anæmia the signs of a pneumohæmothorax. Palpation may detect the apex beat. A splashing sound indicates blood and air in the pericardium. A whizzing sound due to the presence of air in the pericardium is also described. In some cases a friction sound may be heard, and in others a bruit not unlike that heard over an aneurysm. The heart is apt to be irregular and in many cases the pulse is below 100°.

When the bleeding is external the stream may be continuous or in jets. Slight increase in cardiac dulness is to be expected. The respiration may be quiet until near the end, and physical examination of the lung may reveal little abnormal.

When the blood is confined to the pericardium the pulse is exceedingly feeble, and the apex beat can neither be felt nor heard. There may be a splashing sound, disappearing with the filling of the pericardium, at which time the area of precordial dulness will be vastly increased. The patient is unconscious, and may regain his senses on providing an exit for the blood. Cohnheim demonstrated by animal experiments that the pressure manifested itself first on the auricles and the origin of the great veins, producing venous stasis; this may increase to complete obstruction to the flow of blood, and is manifested by dyspnœa and cyanosis. The ventricles pump themselves dry, and the heart finally stops.

Death ensues as the result of anæmia, compression of the heart, or later from sepsis or functional incompetence. Izzo[10] reports a case of stab wound of the left ventricle which was discharged cured on the twenty-eighth day. A few hours later the patient died while lifting a heavy body, the scar having ruptured. The possibility of embolism from air or clot should also be thought of.

As may be seen from the foregoing, a diagnosis of injury of the heart may be difficult or impossible without exploration. The location of the external wound is no sure guide. Of the external wounds in the 60 cases, thirty-nine were on the left side, between the second and seventh ribs, sternum and anterior axillary line, two were in the left midaxillary line, two to the right of the sternum, one in the epigastrium, one under the left costal arch, and fifteen not mentioned. It may be stated, however, that any penetrating wound in the præcordium is almost sure to injure the pericardium or the heart. External bleeding may be profuse and spurting from a wounded intercostal or internal mammary, and may be absent in a punctured wound of the heart. Although justified by Rehn, the use of the probe is generally condemned. The statement that the blood coming from the external wound will be bright red when the left heart is injured and dark in color when coming from the right heart is not to be relied upon. Neither can the size of the wound in the heart be predicted with certainty by the size of the external wound nor the cavity injured by its location. In the present case, for instance, the cardiac wound was larger than the external wound, the knife probably entering the heart at the beginning of systole, and as the heart swung forward added an incised wound to the stab, and the location of the external wound suggested a wound of the auricle rather than the ventricle. Of the 60 cases, the left ventricle was wounded thirty times, with fifteen recoveries; the right ventricle twenty-one times, with seven recoveries; the right auricle once, resulting in death; the left auricle once, resulting in death; the apex three times, with one recovery, and in four the chamber wounded is not known. The size of the wound in the heart muscle varied from 0.5 cm. to 7 cm. The only safe procedure in doubtful cases is to enlarge the wound, ascertain if it penetrates the chest wall, and if there be symptoms of hemorrhage or "heart tamponage," operate.

Although the cases operated upon after the lapse of several hours show a larger proportion of recoveries than those operated upon earlier, operation should be undertaken as quickly as possible after the injury. Of the 60 cases, the time elapsing between the injury and the operation is not given in 29, 11 of which recovered. Of the remaining cases 16 were operated upon within one hour, with 4 recoveries, 1 in three and three-quarter hours with recovery, 4 between three and four hours with 1 recovery, 1 in five hours with recovery, 1 in six hours with recovery, 2 in eight hours with 1 recovery, 2 in twelve hours with 1 recovery, and 3 after twenty-

four hours with 2 recoveries. This gives a mortality of over 76 per cent. for those operated upon within four hours, and a mortality of 30 per cent. for those after four hours. In looking over the causes of death in these cases, and leaving out of consideration 2 cases in which the interventricular septum was cut, it may be noted that of the cases operated on under four hours $35\frac{5}{7}$ per cent. died within twenty-four hours, and of those operated upon after four hours $33\frac{1}{3}$ per cent. died within twenty-four hours. An individual who survives a heart wound for a number of hours has a fair chance to recover with operation, and perhaps without operation, but of the cases which would succumb within a few hours many will be saved by prompt intervention.

The advice given by Rydygier[11] to allow a knife which may be found sticking in the chest to remain until ready to suture is questionable. It is easy to understand how the blade might produce further laceration of a beating heart.

An anæsthetic should always be employed unless the patient be unconscious. Ether was employed in the present case, because it was believed to be safer than chloroform. Of 4 cases in which ether was employed, 2 died, 1 on the table; of 11 cases in which chloroform was used, 4 died, 1 on the table; and of 11 cases in which no anæsthetic was given, 6 died, 2 on the table. Of 2 cases in which it is simply stated that an anæsthetic was administered, both died, 1 on the table.

The heart may be exposed by the extrapleural method or by the transpleural method.

Among those who have employed or who advise the extrapleural method are Rydygier, Pagenstecher, Napalkow, Wehr, Giordano, Podrez, and Nietert. Rydygier[11] advises a horizontal incision at the upper border of the third rib, extending from the right of the sternum to the left costochondral joint, then obliquely downward along the outer ends of the costal cartilages as far as the fifth interspace. The third, fourth, and fifth costochondral articulations are divided, the sternum severed subperiosteally and the flap turned to the right. Giordano suggests a vertical incision along the right margin of the sternum, with the division of the second and third cartilages; from each extremity of the vertical incision a transverse cut is made across the sternum, which is then divided and the flap turned to the left by luxating the cartilages. Podrez begins an incision in the second left interspace one and one-half inches from the sternum, carries it as far as the midsternum, then downward to the level of the seventh cartilage and outward along the seventh rib. The sternum is divided at either end of the longitudinal incision and the flap turned to the left. Nietert[12] cuts from the right border of the sternum to one inch to the left of the sternum on a level with the third rib; a similar incision is made on a level with the base of the ensiform, and the left extremities of these incisions are united by a

perpendicular incision. The fourth, fifth, and sixth ribs and the sternum are divided and reflected to the right.

Among the transpleural methods are those of Del Vecchio, Parlavecchio, Farina, Rehn, Cappelen, Parozzani, Giordano, Ramoni, Ninni, and Rotter. Del Vecchio outlines an H-shaped incision: the vertical limbs correspond to the left sternal and mammary lines, and the horizontal portion to the fourth interspace. The fourth, fifth, and sixth ribs are resected, one flap being turned upward and the other downward. Farina, Rehn, Parlavecchio, and Cappelen resect one or more ribs. Parozzani made a triangular flap, one side of which extended along the left margin of the sternum from the fifth interspace to the ninth rib, the second side from the upper end of this incision outward for 3 cm.; the sixth, seventh, and eighth ribs were cut and the flap turned outward. Giordano incised in the third interspace, sectioned the third and fourth ribs, turning the one upward and the other downward. Ninni advocates an incision along the sternum from the third to the sixth rib and the same length in the mammary line, joining these by a horizontal incision in the fifth interspace. The intervening ribs are cut and the flap turned upward. Rotter cuts outward from the left border of the sternum along the third rib for two inches, then downward parallel with the left border of the sternum to the sixth rib, thence along the sixth rib to the sternum. The third, fourth, and fifth ribs are severed and the flap turned toward the right. He also advises the same procedure on the right side in cases in which the wound is on the right. The opening was made in the right chest in 3 cases only (Watten, Ninni, Barth).

The extrapleural method will be indicated in cases in which the wound does not involve the pleura, and in which the patient's condition is such as to sustain an operation protracted by the small opening in the chest wall, and by the careful manipulations necessary to avoid the pleura. This will be rare; in 3 cases only did the pleura escape injury (Pagenstecher, Nietert, Gibbon). Rehn thinks it advisable to incise the pleura to avoid negative pressure from aspirating air into the mediastinum, causing the blood to foam.

An atypical osteoplastic flap with the base toward the sternum either in the right or left chest, according to indications, and including as many ribs as may be necessary for proper exposure, usually from two to four, will probably be used in most of the future cases.

The wound in the lung ceases to bleed as a rule when the lung collapses and needs no treatment; if bleeding, it may be sutured.

The wound in the pericardium is enlarged in the axis of the heart by tearing or cutting, and the sac cleared of blood with the finger. In several of the cases attention is called to the fact that the blood coagulated behind the heart. In experimental work Ricketts found that collapse of the lung would congest the heart, and that exposure

caused it to dilate because of the lack of support of the pericardium and chest wall.

The bleeding from the heart may be controlled by a finger, and the heart may be steadied by the fingers, by forceps, or by sling sutures. In the present case a suture was inserted at the upper end of the wound, tied, and used as a tractor for the insertion of the remaining stitches. In 2 cases the sutures tore out (Walker, Zulehner). Should the heart cease to beat during the manipulation it should be sutured quickly and massage performed. The use of gauze packing will probably never be indicated; to be efficient it must be firm, and to be firm it must exert injurious compression. A round, curved, intestinal needle is generally recommended. Silk was employed in 26 cases with 2 recoveries, catgut in 9 with 5 recoveries, celluloid in 1 with recovery, and in 24 the suture material is not known. The highest number of sutures employed was eleven. Silk has the advantage of being easier to manipulate and more certain to hold. It has been proven that catgut will last long enough for the repair of the wound. Silk was used in the present case because of the fear of untying of the knots by the constant gyrations of the heart. The continuous suture is mentioned in 3 of the cases with 2 recoveries. It may be applied more rapidly than the interrupted, and presents fewer knots on the surface of the heart. The sutures are inserted at intervals of one-eighth of an inch deeply into the muscle, but must not penetrate the endocardium. In dogs Sherman[4] found it a difficult matter to penetrate the endocardium with an ordinary curved needle. After tying, the ends should be cut off one-quarter of an inch from the knot to guard against untwisting. Diastolic suture is difficult and unnecessary. One would have to rehearse with a rapidly ticking metronome for a long time before he would be able to accomplish the feat. In dogs Ricketts[14] and Sherman[4] found it impossible. Although Larrey showed many years ago the possibility of recovery after a severed coronary artery, it has been asserted repeatedly that this accident is always fatal. Ricketts demonstrated on animals that either coronary artery may be ligated at any point without death resulting. Of the 4 cases in which the coronary was injured, only 1, the present case, recovered.

Any blood which may be in the pericardium is now removed by the finger or sponge, and the pleural cavity cleared of fluid and clots. The pleura and pericardium may be sutured or both cavities may be drained. Wolff[8] shows that there is no retention of secretion in the pericardium when it is closed. He explains the presence of fluid behind a tampon in Watten's case by the massage of the heart by the gauze. He says the cardiac wound heals quickly and is not infected, the instrument having been cleaned on its way through the chest wall, and he strongly advises suture of the pericardium without drainage. The dangers of the drain are the same

here as elsewhere—*i. e.*, infection and adhesions. Of the 22 cases in which the pericardium was drained, 2 died within a few hours, and of the remaining 20, 10 (50 per cent.) recovered; of the 15 in which the pericardium was closed without drainage, 1 died in fifteen minutes; and of the remaining 14, 7 (50 per cent.) recovered. The question may still be regarded as an open one. In a future case I think I should close the pericardium and drain the pleura through a stab in the back, as the latter cavity is difficult to clear of blood quickly.

Of the 23 cases that recovered after operation 11 are known to have been complicated with some form of infection; in only 1 case is uncomplicated recovery stated. Of the 37 deaths, 16 occurred within twenty-four hours from shock and hemorrhage, and in 2 of these the interventricular septum had been punctured; 3 died on the second day, 2 from the infection of the pleura and 1 unknown; 4 died on the third day, 2 from infection and 2 unknown; 2 died on the fourth day, both from infection, and in 1 of these the tricuspid valve had been injured; 3 died on the fifth day from infection, and 1 on the sixth, 1 on the fourteenth, 1 on the nineteenth, and 1 on the twenty-second day, all from infection; in 5 cases the time and cause of death are unknown.

I append a table of the reported cases. (See pages 442, 443.)

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Table of Cases of Cardiorrhaphy.

Operator and year.	Location of external wound.	Chamber wounded and size of wound.	Time between injury and operat'n.	Anæsthetic.	Suture.	Drainage	Result and remarks.
1. Farina, 1896	Just above margin of left 6th rib, near sternum.	R. v., ¼ in.	Immediately.	.....	3 silk sutures.	Suture of pericardium.	Death 6th day; bronchopneumonia.
2. Cappelen, 1896	4th left intercostal space in midaxillary line.	L. v., ⅘ in.; left coronary artery sever'd	1 hour.	.....	.....	.....	Death 2½ days; pericarditis.
3. Rehn, 1897	4th left intercostal space near sternum.	R. v., 1½ cm.	24 hours.	Ether.	3 silk stitches	Drain of pleura and pericard'm	Recovery; empyema.
4. Parozzani 1897	7th left intercostal space in midaxillary line.	L. v., ¾ in.	5 hours.	None.	2 silk sutures.	None.	Recovery.
5. Parozzani 1897	3d left intercostal space.	L. v., ⅘ in.	½ hour.	None.	2 silk sutures.	....	Death 24 hrs.; interventricular septum cut, also pleurisy on l. side.
6. Fummi, 1898	Under left nipple.	Apex, cavity not opened.	Several hours.	.....	1 silk suture.	.....	Recovery; empyema.
7. Ninni, 1898	5th left intercostal space, mammary line.	L. v., 1 in.	Immediately.	None.	2 silk sutures.	No drain pericard'm.	Death on table.
8. Parlavecchio, 1898	5th left intercostal space.	L. v., 1½ in.	8 hours.	Chloroform.	.....	No drain.	Recovery.
9. Giordano, 1898	2d left intercostal space.	L. a., ⅘ in.	½ hour.	None.	4 silk sutures.	Drain of pleura.	Death 19th day; empyema and abscess r. lung.
10. Williams, 1898	.....	L. v., not penetrating.	.....	.....	.....	.....	Recovery.
11. Nicolai, 1899	4th left intercostal space, mid between sternum & nipple.	R. v.	1½ hrs.	Yes.	.....	.....	Death after 12 hours.
12. Tuzzi, 1899	4th left intercostal space.	2 wounds, non-penetrating.	.....	None.	.....	Drain of pleura and pericard'm.	Death 22 days; empyema and pericarditis.
13. Longo, 1899	5th left intercostal space ⅖ in. internal to nipple.	L. v.	Immediately.	None.	3 silk sutures.	No drain of pericard'm.	Death in 15 min.
14. Ramoni, 1899	3d left costal cartilage ⅘ inch from sternum	R. v., 2 w'ds, 1 non-penetrating.	.....	None.	4 silk sutures.	No drain of pericard'm, pleura dr.	Recovery.
15. Marion, 1899	Shot through breast.	R. v.	.....	.....	Catgut sutures.	.....	Death.
16. Rosa, 1899	5th intercostal space.	L. v., ⅗ in. not certain it penetrat'd	.....	None.	1 silk suture.	Drain of pleura and pericard'm.	Recovery.
17. Horodynski, 1899	.....	R. v., 1¼ cm.	.....	.....	.....	.....	Death.
18. Maliszewski, 1899	....	.....	.....	.....	.....	Drain of pericard'm.	Death; suppurative pericarditis.
19. Maliszewski, 1899	.....	.....	.....	.....	.....	Drain of pericard'm.	Death; suppurative pericarditis.
20. Bufnoir, 1899	6th left intercostal space.	R. v., gunshot, 22 calibre.	.....	... ..	.....	.....	Death; perfor'n of ventricle, and aut. opening only had been sutured
21. Kosinki, 1899	.....	R. v.	.....	.....	.....	.....	Recovery.
22. Pagenstecher, 1899	4th left intercostal space beneath nipple.	L. v., near apex, 1⅖ in.	2 days.	None.	4 celluloid sutures.	Drain by threads from myocardium.	Recovery.
23. Nanu, 1900	3d intercostal space 4 cm. from edge of sternum.	R. v., 2 cm	.....	.....	2 interrupted sutures of silk.	.....	Death 5th day from infection of pericardium and pleura.
24. Masselli, 1900	Below and internal to left nipple, cutting 6th rib.	L. v., near apex.	1½ hrs.	None.	2 sutures	Drain of pleura.	Death in 12 hrs.
25. Fontan, 1900	6 wounds between 3d and 7th ribs in cardiac region.	L. v., 12 mm.	6 hours.	Chloroform.	2 continuous catgut sutures.	No drain, suture of pericard'm.	Recovery.
26. Carnoble, 1900	.....	Apex.	.....	.....	Catgut.	.....	Death in 5 days; empyema.
27. Nietert, 1901	Right of sternum.	R. v.	2 hours.	None, unconscious.	2 silk sutures.	Drain of pericard'm.	Death in 33 hrs.
28. Vaughan, 1901	5th left costal cartilage divided.	L. v., 2½ cm.	¾ hour.	Ether.	7 continuous silk sutures.	.....	Death on table.
29. Ninni, 1901	Left of sternum.	R. a.	.....	.....	.....	.....	Death in 4 days; sepsis.
30. Mignon et Sieur, 1901	.....	R. v.	.....	.....	.....	.....	Death.

Operator and year.	Location of external wound.	Chamber wounded and size of wound.	Time between injury and operat'n	Anæsthetic.	Suture.	Drainage	Result and remarks.
31. Fontan, 1901	.....	L. v.	.....	.....	Catgut sutures.	Suture of pericard'm.	Recovery; empyema.
32. Brenner, 1901	Left of sternum near 6th cartilage.	R. v., 7 cm.	Following day.	Yes.	.....	.....	Death on table.
33. Watten, 1901	4th right intercostal space.	R. v., 3½ to 4 cm.	.....	.....	.....	Drain of pericard'm.	Recovery; right pleura wounded, pneumothorax.
34. Lastaria, 1901	.....	L. v.	.....	.....	.....	.....	Death in 12 hrs.
35. Zulehner, 1901	.....	L. v., ½ cm.	.....	.....	.....	.. ..	Death on table.
36. Zulehner, 1901	.....	R. v. 1 cm.	.....	.....	Catgut sutures.	.....	Recovery.
37. Stern, 1901	.....	L. v., pistol wound.	.....	.....	Unable to suture posterior wound.	Gauze tampon.	Death in 30 min.
38. Pagenstecher, 1901	In heart region.	L. v., left coronary artery injured.	.....	.....	.....	Drain of pericard'm.	Death in 5 days; pus in pleura and pericard'm.
39. Ninni, 1901	.....	L. v.	.....	.....	.....	.....	Recovery.
40. Walker, 1901	.....	L. v., 8 stab wounds.	.....	.....	11 silk sutures.	Drain of pericard'm.	Death in 24 hrs.; pyothorax and pyopericardium.
41. Walker, 1901	Under left costal margin.	Apex r. v.	.....	.....	Suture tore out repeatedly.	Gauze tampon.	Death in ½ hr.; hemorrhage.
42. Nietert, 1902	Left 6th intercostal space to right of papillary line.	L. v., 2 cm.	14½ hrs	Chloroform.	2 silk sutures.	Drain.	Recovery; empyema.
43. Launay, 1902	Left nipple.	L. v., ant. & post. walls, pistol shot.	3½ hrs.	Chloroform.	Catgut suture in each wound.	Drain of both cavities.	Recovery; uncomplicated.
44. Gibbon, 1902	Left præcordium.	R.v., 1.5 c.m.	½ hour.	Chloroform.	1 catgut suture.	.....	Death on table.
45. Hill, 1902	5th intercostal space a little to right of nipple.	L. v., ⅝ in.	8 hours.	Chloroform.	1 catgut suture.	.....	Recovery.
46. Stewart, 1902	4th intercostal space ½ inch left of sternum.	L. v., ⅝ in.	Immediately.	Ether.	6 catgut sutures.	.....	Death in 3 days.
47. Pomara,[15] 1902	.....	.....	.....	.....	.....	.....	Death in a few days.
48. Milesi,[16] 1903	Upper border 6th rib 1.5 cm. from left border of sternum.	R.v., 1.5 c.m.	½ hour.	.....	Silk suture.	Drain of pleura and pericard'm.	Death in 15 hrs.; punct. interventricular septum.
49. Giordano[17] 1903	2 wounds, 1 in 4th and 1 in 6th intercostal space.	L. v., 2 cm. long.	.....	.....	2 silk sutures.	Drain.	Recovery; necrosis of ribs.
50. Giordano[17] 1903	6th intercostal space 1 cm. left of nipple.	L. v., near apex.	.....	.....	2 silk sutures.	Drain.	Death in 1½ hrs.
51. Vince,[18] 1903	5th left intercostal space in parasternal line.	L. v., 6 cm., coronary artery injur'd during suturing.	About 12 hours.	Chloroform.	3 silk sutures.	No drain of pericardium, drain of pleura.	Death 2d day; pneumonia and pleurisy.
52. Janari,[19] 1903	3d left intercostal space bet. ant. axillary & mam. lines.	L. v., 2 cm.	¼ hour.	Chloroform.	3 silk sutures.	Drain of pericard'm.	Death in 74 hrs.; pleurisy.
53. Barth, 1903	Epigastrium and through ensiform.	R v., 2 cm.	Soon.	Chloroform.	4 silk sutures.	Drain of pleura and pericard'm.	Death 4th day; sepsis tricuspid valve injured.
54. Barth, 1903	4th left intercostal space 3 cm. from sternum.	L. v., ½ cm.	Soon.	Chloroform.	3 silk sutures.	Suture of pericard'm.	Recovery, with pleurisy.
55. Barth, 1903	3 cm. above and inside nipple.	R. v., ¼ cm.	.....	Chloroform.	5 silk sutures.	Suture of pericard'm.	Recovery, with pleurisy.
56. Schwerin[20] 1903	4th left intercostal space.	R. v.	¼ hour.	.....	3 silk sutures.	Drain.	Recovery; pyopericardium.
57. Noll,[21] 1903	Revolver wound 5th left intercostal space.	L.v., ant. wall sutur'd, post. not ment'n'd.	1½ hour.	.....	.....	Drain.	Recovery; empyema.
58. Wolf,[22] 1903	.....	R. v., 2 cm.	.....	.....	.....	Drain.	Death in 14 days; empyema.
59. Stude,[23] 1904	Left breast.	L. v., 1 cm.	1¼ hour.	.....	Silk suture.	.....	Death on table.
60. F. T. Stewart, 1904	2d intercostal space 1 inch left of sternum.	L. v., ¾ in., coronary artery injured during suturing.	¾ hour.	Ether.	6 continuous silk sutures.	Drain.	Recovery; pneumonia and infection of wound in chest wall.

Cases 1 to 46 inclusive and Cases 58, 54, and 55 are taken from Hill[10] and Wolff.[8]

THE TREATMENT OF PENETRATING WOUNDS OF THE HEART.[1]

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It is difficult for anyone to speak with authority born of experience on the treatment of penetrating wounds of the heart, since opportunity for repair of such injuries is so rare. Dr. Stewart has presented a table of 60 cases, which are all that have been recorded in surgical literature, and but two surgeons have operated on as many as three cases. My own excuse for attempting to discuss this most important subject is the interest I take in it, due to an unsuccessful effort to suture a stab wound of the right ventricle. I have reported this case in the *Philadelphia Medical Journal*, November 1, 1902. This was the fourth case operated upon in this country.

Dr. John B. Roberts was one of the first to suggest the advisability of suture in cases of penetrating wounds of the heart. This he did in a paper read before the Anatomical and Surgical Society at Brooklyn in 1881. It was, however, not until 1896 that the first case of operation for this condition was reported. Dr. Stewart's table, perhaps, does not present all the operations, since no doubt there are a number of unsuccessful cases which have never been put on record. The results obtained in these cases, twenty-three recoveries, certainly teach us that the former surgical attitude toward penetrating wounds of the heart was altogether wrong, and that we should always attempt their repair whenever the opportunity presents itself. It is evident that certain cases are more hopeful than others. Those patients who are able to survive the injury for some hours or even a day are much more likely to recover after operation than those who immediately pass into unconsciousness and present other symptoms of cardiac compression or those of excessive loss of blood. The repair of gunshot wounds of the heart has not been so successful as that of stab wounds, and probably one reason for this is the fact that in the former there are often two wounds. Peyrot says that 17 per cent. of gunshot wounds are double, while only 1.2 per cent. of stab wounds are. In this connection it is interesting to refer to a case which Trendelenburg reports, in which a bullet was lodged in the right ventricle and the patient recovered without operation. The bullet could be seen with the fluoroscope and its movements observed. Later, it became fixed in the heart wall. Strange as it may seem, there are 19 cases

[1] Read before the College of Physicians of Philadelphia, May 4, 1904.

on record where bullets have lodged either in the heart cavities or wall and the patients lived for varying periods. Rickets, in his most exhaustive study of the surgery of the heart, refers to the frequency with which the heart has been found to be most unexpectedly the seat of injury due to its anomalous situation upon the right side or even within the abdomen.

As one would expect to find, the Italian surgeons are those who have contributed most largely to the literature on this subject. The Germans also have reported a large number of cases. Williams, of Chicago, in 1898, reported the first successful case in America. Nietert, of St. Louis, in 1901, lost a patient thirty-three hours after a successful suturing of the heart from suppression of urine, but was able a year later to report a successful case, the second in this country. Hill, of Birmingham, also has recently reported a recovery after operation. The case reported by Stewart is, therefore, but the fourth successful one operated upon in America.

Regarding the technique of repairing penetrating wounds of the heart, it is impossible to lay down any fixed rule, since it must vary largely with the general condition of the patient and the situation of the wound. In my own unsuccessful case I think I lost valuable time in scrubbing the field of operation after the patient was put upon the table, but I should have felt very remorseful had he recovered from his injury and died from sepsis. From a study of the reported cases, however, I would conclude that in patients who are in a desperate condition the part of wisdom would be to expose and repair the heart with the least possible delay. In patients, on the contrary, who have survived the injury for a number of hours or who are not in an immediately dangerous condition, sufficient time should be taken to thoroughly disinfect the field of operation. The question of an anæsthetic is one about which there has been considerable discussion, and here again it is not possible to form any fixed rule, since in the completely unconscious cases an anæsthetic is unnecessary, and in the conscious cases it is as strongly indicated. That the manipulation and suture of the heart itself is not painful is shown in Nietert's first case, where the patient regained consciousness during the introduction of the sutures and conversed with the surgeon during the closure of the wound. Although an anæsthetic has its disadvantages at such a time, the struggling resulting from the exposure of the heart without an anæsthetic carries with it the danger of loosening a clot which may have temporarily occluded the opening in the cardiac wall. This danger can be illustrated in a case of penetrating wound of the heart which healed without operation, and in which the patient died some time later after violent exertion, producing a dislodgement of the hæmostatic clot. The incision and the nature of the flap must vary with the seat of the injury and also with the promptness with which the heart must be exposed. The tendency of most

operators in their first cases has been to resect the necessary amount of bone and cartilage to thoroughly expose the wound. It is the judgment of the most experienced, however, that a deliberate osteoplastic flap with its base at the sternum or beyond the costo-cartilaginous articulations is to be preferred. Having found the wound in the pericardium and opened it, if bleeding is taking place, a hurried digital examination of the heart should be made, and when the opening is found the tip of the finger should be inserted into it and carefully kept in position in order to prevent the escape of more blood. In some instances it has been possible to introduce the sutures while the finger was still inserted in the opening. If this cannot be done two procedures may be followed: that of introducing traction sutures for the purpose of lifting up the heart, and the other, of placing two fingers behind the heart and so bringing it into the wound. Suture during diastole has been strongly recommended by some. Those, however, who have had experience in closing such wounds seem to agree that it makes little difference when the suture is introduced. It seems equally unnecessary to carefully avoid entering the heart cavity with the suture, since a number of experimenters have shown that it is no easy matter to introduce the suture to that depth. It makes little difference what suture material is used, although personally I should prefer chromicized gut introduced on a small, full-curved intestinal needle. When catgut is used, however, care must be taken to make at least three knots, so as to overcome the danger of loosening. Having closed the anterior heart wound a rapid search should be made for a wound of exit; if none is found the pericardium should then be cleared of clots by damp sponging and the pericardial wound closed with or without drainage. If there has been a wound of the pleura, as in most instances there is, the blood should be removed from this cavity and its wound closed with or without drainage. If the pleura has not been involved in the primary injury it is apt to be opened in the exposure of the heart. The question of drainage is one about which there is also considerable dispute. If the operator has been able to thoroughly cleanse the field of operation, and believes that there has been no contamination during the operation, I can see no more reason to drain either the pericardium or the pleura than there would be after opening the abdomen for an intra-abdominal hemorrhage. If, on the contrary, the operation has been done hurriedly and without thorough preparation, then I think that drainage of both the pericardial and pleural cavities should be established. If it should be desired to drain the pleura and not the pericardium, I believe that this had best be done at a point distant from the field of operation, since in this way drainage at the most dependent point can be obtained, and, in case suppuration takes place, infection of the pericardium possibly avoided.

MEDICAL AND SURGICAL CONSIDERATIONS IN PYOPERICARDITIS, WITH REPORT OF CASES.[1]

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MEDICAL ASPECTS, BY J. A. SCOTT, M.D.

Pericarditis is usually divided into primary and secondary varieties. Primary pericarditis is so rare that it can be dismissed without further consideration. Secondary pericarditis, or that concurrent with or sequent to other diseases, is that commonly met with in practice. This paper deals only with purulent pericarditis of this type.

Frequency of Pericarditis. This is a difficult matter to decide, for statistics vary considerably. Anders' figures, collected to show the frequency of cardiovascular lesions in croupous pneumonia, show in 275 autopsies the presence of acute pericarditis in 14 cases, or 5 per cent., and chronic pericarditis in 11 cases, or 4 per cent. Riesman, in a study of adhesive pericarditis, analyzed 778 necropsies in the Philadelphia Hospital and found 60 cases of pericarditis, 7.7 per cent.; 20 of these were adhesive; very few were purulent. Breitung, collecting 324 cases of pericarditis at the Berlin Charité, found the following figures: serofibrinous cases, 108; hemorrhagic, 30; purulent, 20 (6.1 per cent.); secondary tuberculous, 24; primary tuberculous, 2; partially adhesive, 111; totally adhesive, 23; ossifying, 2. The proportion of cases of pericarditis occurring in autopsy work is given in von Ziemssen's *Handbook* by various authorities as follows: Duchet, 15.1 per cent.; Chamber, 16.2 per cent.; Willigh, 14.1 per cent.; Taylor, 12.5 per cent. G. E. Still, in an article on "Suppurative Pericarditis in Children," finds in 769 autopsies 28 cases of pericarditis, apparently due to pyogenic infection. Twenty-three of the 28 cases occurred in children under three years of age, and 24 of the 28 showed empyema, thick lymph on the pleura, or acute pleurisy. In 13 cases in which the fluid was examined bacteriologically, 11 were due to pneumococcal infection; of the remaining 15, a large proportion were due to the pneumococcus—empyema, pneumonia, and meningitis being associated.

[1] Read before the College of Physicians of Philadelphia, May 4, 1904.

Analyzing the mortality statistics of the Pennsylvania Hospital since the opening of the Ayer Clinical Laboratory, I have found 40 cases of pericarditis in 552 autopsies. Ten were obliterative in type; in 13 the exudate was serofibrinous; in 16 (40 per cent.), purulent; in 1 hemorrhagic. The great discrepancy will be observed at once between these statistics and those of Breitung, in which were found but 20 cases of purulent pericarditis in a total of 324 cases of all varieties. Clinically these 40 cases were etiologically as follows: tuberculous, secondary, 7; rheumatic, with acute or chronic endocarditis, 5; aneurysm, cardiac and abdominal, 2; nephritis, general streptococcus septicæmia, cirrhosis of liver, and secondary anæmia, 1 each; and lobar pneumonia, 22. In all these 22 cases pleurisy coexisted with the pericarditis; in 6 cases both the pericarditis and pleurisy were fibrinopurulent; in 4 each process was fibrinous; in 3 cases purulent pericarditis was associated with plastic pleurisy, and in 3 cases the pleurisy was fibrinopurulent, while the pericarditis was fibrinous. In 2 cases plastic pleurisy and adhesive pericarditis coexisted, and in 2 others the pericarditis was purulent, while the pleurisy was serofibrinous. In the remaining case the pericarditis was serofibrinous, and the pleural effusion was bloody.

Again, recognizing the great frequency of purulent pericarditis in croupous pneumonia, I have gone over the total number of autopsies upon that disease and find them to total 76 cases of croupous pneumonia and 5 of bronchopneumonia. In 38 cases of croupous pneumonia, or exactly 50 per cent., pericarditis has been found; in 20 cases, or 52.6 per cent., the exudate was serofibrinous; in 17, or 44.7 per cent., the pericardium contained a purulent exudate; in one case the pericardium was obliterated (chronic adhesive pericarditis). In none of the bronchopneumonias (non-tubercular in origin) was pericarditis present.

Etiology. Purulent pericarditis is seen in pyæmic diseases—the streptococcal and staphylococcal infections—as a result of empyema, and in perforation of abscesses in the neighborhood of the pericardium; cancer of the œsophagus, cardiac and aortic aneurysm, etc. It is, I believe, commoner during lobar pneumonia than is recognized, and, as the autopsy records at the Pennsylvania Hospital will show, must be responsible for the death of the patient in not a few cases. The filling of the pericardium with exudate during croupous pneumonia is, I believe, not infrequently overlooked. The diagnosis, however, under such circumstances is a difficult one, as will be detailed later.

Bacteriology. The variety of micro-organisms found in purulent pericarditis depends upon the primary infection producing the disease. The purulent form is most commonly caused by streptococci, staphylococci, bacilli coli communis, pneumococci, and the tubercle bacilli. There is no reason why the gonococcus and the typhoid bacillus should not play a part in purulent pericarditis.

When a connection is established between the pericardium and the œsophagus or the lung numerous saprophytic micro-organisms may be found.

SYMPTOMATOLOGY. Symptoms are often difficult to interpret correctly, mainly because of the fact that pyopericarditis is commonly only one part of the symptomatology of such serious illnesses as a general pyæmic state, abscesses of the mediastinum, or pneumococcal and other infections.

General Appearance. The patient usually presents an anxious expression and dusky pallor of the skin, with dilatation of the alæ of the nose. Dyspnœa is usually present, at times extreme. The pulse in these cases is quick, with a sharp rise under the finger, rapid and of low tension; very unlike the pulse of an acute infection. At times the large veins of the neck are much distended.

Pain. Common usually over the region of the heart, but of moderate severity; again, it may be intense and agonizing, radiating over the left chest and down the left arm. It is commonly increased by pressure in the epigastric region. During the development of a purulent pericarditis, and while friction rub is present, the pain is apt to be more severe and disappears to some extent with the presence of a large effusion. In many cases pain in the epigastric region is an important symptom; this is accompanied with a sense of constant fulness in the same area; nausea and vomiting are apt to be present in this type. The vomiting is bile-tinged in some cases.

Dyspnœa is usually present and is disproportionate to the amount of pulmonary involvement and the rate of pulse.

Cough. Often dry, frequent, and distressing. Often non-productive. If the pericarditis is associated with pneumonia the sputum may, of course, be blood-tinged. Aphonia and dysphagia may be present.

Temperature. Fever is, of course, complained of to some extent. The temperature is at times but slightly elevated, though the pericardium may be filled with pus. The pulse is weak, small, and may present an intermittency known as the pulsus paradoxus, a condition in which the radial is either greatly weakened or altogether obliterated during inspiration. Delirium in pericarditis of all forms is common. At times it is active, at other times it is low. It is especially to be found in the rheumatic cases with hyperpyrexia.

PHYSICAL SIGNS. Purulent pericarditis at times develops without the ordinary physical signs of the disease being present, or at least discoverable; that is, though carefully sought for, the presence of friction rubs over any portion of the precordial area cannot be detected. In many cases pericardial friction rubs antedate the presence of effusion. This is certainly so in the serofibrinous cases. They are heard most commonly in the second, third, and fourth interspaces to the left of the sternum, perhaps more frequently over

the base of the heart than at the apex. They are close to the ear, rough in character; sometimes both coarse and fine sounds can be heard. Heard with the systole and diastole of the heart and not always consistent in length or duration. Pressure increases their intensity. Their inconsistency is their characteristic. Change in position alters the character and time of the sound. At times these rubs can be heard over a large area of the chest; under some circumstances as far as each nipple and down into the epigastric region. Friction sounds, of course, disappear with the presence of effusion into the pericardial sac. Under such circumstances a distinct alteration in physical signs occurs. The apex beat, which heretofore was in its normal position, becomes slightly elevated, perhaps to the fourth space, or may disappear altogether. It may, however, still be palpable in the fourth space. The sounds at the apex are distant and weak. With a normal endocardium no valvular murmurs are heard. Frequently, however, in these cases the causative factor in producing the pericarditis also has caused an endocarditis. Not infrequently the two conditions coexist. Valvular murmurs are therefore to be heard at the areas affected. Coincident with the disappearance of the apex beat occurs an increase in the precordial area of dulness. This is especially noticeable upward to the left of the sternum, perhaps to the second rib and in the fifth and fourth spaces to the right of the sternum. Impaired resonance or dulness discovered in the fifth or fourth interspaces to the right of the sternum, with decided increase of the left border of the cardiac outline and disappearance of the apex beat with weak or feeble heart sounds, is certainly extremely suspicious of fluid in the pericardium. Under such circumstances other physical signs are apt to be present. Owing to the presence of large quantities of fluid in the pericardial sac there is compression of the left lung, especially the lower lobe, in the axillary region. Here at times can be detected a hyperresonant note. The same pressure frequently will produce an impaired or dull note at the extreme base of the left lung. On auscultation at this point the breath sounds may be bronchovesicular or bronchial. This is a pressure consolidation. With physical signs such as these no hesitancy should be felt in inserting a needle into the fourth or fifth space near the left sternum, especially if the absence of the apex beat is demonstrated. In not a few cases fluid will be secured by exploratory puncture in the fourth or fifth right intercostal space. In some cases, however, the physical signs are extremely difficult of interpretation, owing to the presence of complicating conditions. Thus not infrequently in croupous pneumonia there is fluid both in the chest and in the pericardium. Thus in Case II. an empyema of very considerable size dislocated the heart to the left so that the impulse was visible in the fifth interspace one inch outside the midclavicular line. With the evacuation of the empyema the pericar-

dial condition was entirely overlooked, and this probably resulted in the man's demise. In two other cases under observation (Case IV. and one not reported in this paper) there was visible pulsation in the second and third interspaces to the right of the sternum, with the development of (in Case IV.) an enormous shield-like area of dulness between the two nipples, with absolutely clear breath sounds at the bases and axillary region on both sides. These physical signs could only therefore be explained by the supposition that the right side of the heart was adherent to the pericardium or that its pericardial effusion had displaced the heart *in toto* to the right. In the unreported case spoken of, adhesions between the lung and the pleura on the left side were so dense that the presumption is that the suspected pericardial effusion was behind both the lung and the heart, which in turn was displaced considerably to the right.

TEMPERATURE. The range of fever in purulent pericarditis is less than one would anticipate. As previously stated, the presence of pus in the pericardium will at times distinctly modify an already pre-existing high temperature. In the first case studied, while the pericardium was filled with the purulent exudate, the temperature was subnormal for a week. In Cases II. and III. the temperature range with the pus in the pericardium was between 98° and 100° F., and 100° and 102° F., respectively. In Case IV. the temperature was high. It is difficult to state the exact time of the onset of the pericarditis.

PULSE. The pulse is weak and of low tension from the start, irregular at times; in some cases influenced by respiration, either partially or totally obliterated. The paradoxical pulse was present in Case I.

BLOOD. A leukocytosis is produced in all cases. If, however, the pus has been present for any length of time in the pericardium, the leukocytes have a tendency to fall again toward, but do not reach, the normal. In cases with pre-existing leukocytosis—and this is found in practically all patients with purulent pericarditis—a hyperleukocytosis is added. In Case I. the leukocytes jumped from 6600 on the tenth day to 21,000 on the sixteenth day, and fell slowly to 17,050 on the twenty-eighth day. Three blood cultures were negative. In Case II. a leukocytosis of 22,700 was present on the fourteenth day. The pericardium at that time was probably full. On the twentieth day with both pericarditis and empyema present the leukocytes were 12,000. The blood was not counted after operation; no blood cultures were taken. In Case III. the leukocytes on the sixth day were 16,400; on the tenth day, 30,800. At this time pericarditis was diagnosed. Blood cultures on the third and fifth day were negative, but a positive culture was found on the tenth day after pericarditis was present. In Case IV. no cultures or counts were made. The urine is much reduced in quantity in all cases; usually contains albumin, no sugar, and on microscopic

examination shows rather numerous casts; in our case granular casts have predominated, though hyaline were also found. The urea output in Case II. (the fatal case) was 30 grm. in the twenty-four hours.

In conclusion, in pneumonia the cardiac outline should be carefully mapped out at the onset. Any changes either in the size or position of the heart should carefully be watched for. With the development of an empyema more rigid scrutiny of the heart should be insisted upon. The presence of dulness in the fifth right interspace or the dislocation of the heart either to the right or to the left side without a coexisting empyema, should lead one to suspect the presence of fluid in the pericardium, and careful selection of the spot for exploratory puncture should be made.

Diagnosis. Pyopericarditis is to be diagnosed by the following signs: The presence of a primary infection capable of producing pus; the development of a rapid, weak, and at times intermittent pulse, cough, and dyspnœa; pain over the heart and epigastric region of severe nature; a temperature of mild degree, and mental symptoms of varying intensity. Associated with these symptoms pericardial friction rubs over the base or the entire cardiac area may be heard. They are at times altogether absent (Cases I. and II.); then follows the rapidly increasing, pear-shaped precordial area of dulness, extending upward to the left second rib, to the right in the fifth, fourth, and third intercostal spaces, at times reaching the right midclavicular line, and to the left to and beyond the left midclavicular line. It is important to note that the apex beat, if present, is within this area of dulness and not at its extreme limits. The shape of this dull area varies with the amount of fluid in the sac. If the quantity is not large the dull area is pear-shaped with its base exaggerated. If the quantity is large the dulness is almost circular or ovoid at its upper border, the centre of the bow being the sternum. The lower dulness merges with that of the liver or even the spleen, but more often with the dulness or flatness of the consolidated lung or that produced by fluid in the pleura. The apex is either lifted to the fourth space or disappears altogether, or the entire heart is bodily displaced to the right or left, according to circumstances.

The sounds at the apex grow distant and faint and at times can be heard with difficulty. The left axilla presents a hyperresonant note with exaggerated breathing; the left base may show a spot of compression dulness with bronchial breathing. To clinch the diagnosis an exploratory puncture should be made in that portion of the dull area farthest away from the ascertained position of the heart, preferably in the fourth or fifth space to the left of the sternum.

Differential Diagnosis. 1. Serofibrinous pericarditis. 2. Acute dilatation of the heart. 3. Acute catarrhal gastritis. 4. Right-sided or left-sided pleural effusion or empyema, either free or loculated. Some unusual conditions have been mistaken for pericar-

ditis, such as mediastinal tumor, acute endocarditis, cases of aortic regurgitation, and we have recently heard that perforation of the bowel was diagnosed by two very competent observers, the man's condition, however, preventing operation, and the autopsy showing the presence of a purulent pericarditis.

Before considering any of these points in diagnosis the following anatomical belief usually stated in text-books is questioned: That the heart usually or always, in the presence of large quantities of fluid in the pericardium, is pushed away from the chest wall, with the consequent disappearance of the apex beat and weakness of sounds. The heart is fixed at its base by the attachment of its vessels, and this leaves the apex and the ventricles as the movable portion to be effected by pressure from all sides or any side; if, however, adhesions already exist between the pericardium and the heart on either the right or left side a lateral dislocation of the heart will occur; or pressure exerted upon the filled pericardium as a whole may cause a lateral dislocation of the heart; perhaps other unknown conditions within the pericardium may produce the same lateral displacement. We hope in the future to be able, with the kind assistance of Dr. Longcope, to experimentally verify some of these views; as yet we have not been able to do so.

1. Serofibrinous pericarditis is usually the result of rheumatic infection or concurrent with tuberculosis or Bright's disease. The history is of decided importance as the causative factor. The symptoms and signs are, of course, much the same as in purulent pericarditis. You have not, however, the usual chest complications seen in the purulent variety. The temperature is apt to be but slight, and the leukocytosis does not reach such a grade as in the purulent form. This fact, however, is not to be relied upon. Cardiac dilatation is common in the rheumatic pericarditis and endocarditis frequent. Dilatation of the heart is not common in purulent pericarditis. The physical signs in both conditions are similar except that we must recall the fact that purulent pericarditis is seldom present without a purulent pleurisy, a pneumonia, or meningitis, or pus or ulceration near the pericardium. Exploratory puncture settles the doubt at once.

2. Acute dilatation of the heart is a common feature of chronic valvular disease and rheumatic pericarditis, but according to Still is uncommon in purulent pericarditis. It is attended with weak, rapid, and irregular pulse. The cardiac impulse, however, is visible, wavy, and occupies several interspaces, although at times it is not palpable; the impulse may be irregular, now weak and now strong, and can be perceived in the epigastric region. The heart sounds are heard, the first short, sharp, but weak, and not either distant or absent. There is a decided increase in the area of cardiac dulness, which, however, does not extend so high as in pyopericarditis. The second sound is sometimes split with so-called "gallop rhythm."

Careful mapping out of the heart dulness shows the mobility of the heart. In dilatation there is a tendency to venous congestion and dropsy; the liver increases in size, the kidneys fail to secrete, and the lungs fill up. The *x*-ray examination is a decided help, showing in dilatation a fixed quadrangular shadow; in pyopericarditis a movable upper line of dulness. The blood count will show the absence of leukocytosis in dilatation. When dilatation of the heart occurs in rheumatic pericarditis in a case in which friction rubs have been easily demonstrable, the diagnosis becomes extremely difficult. It rests mainly on the points mentioned above, yet we have seen the diagnosis of acute pericardial effusion with request for immediate opening of the pericardium made by a most able diagnostician, and the operation show an acute cardiac dilatation.

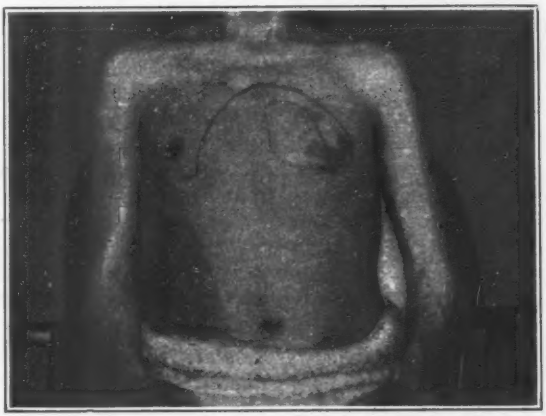
3. Acute catarrhal gastritis presents the history of some causative factor, such as the ingestion of indigestible food, drink, or irritating substance. The vomiting is more or less constant and the pain confined to the gastric area. The temperature is usually high and cough and dyspnœa are not present. The cardiac outline is normal in size and the heart sounds well heard. The blood is of little help, as a leukocytosis may be present in acute gastritis.

4. Pleural effusion, either free or loculated. It is taken for granted that the reader is familiar with the signs of free fluid, be it either serum or pus, in the chest. When this is present in sufficient quantity so that the lower limit of dulness reaches the anterolateral portions of the chest and impinges upon the normal heart dulness, we have a condition which presents many difficulties in diagnosis. If the case has been seen from the start and the size of the heart and the character of its sounds carefully noted, the diagnosis is perhaps not difficult, but when brought face to face for the first time with the presence of large quantities of fluid in the chest with the dislocation of the heart either to the right or to the left and apparent increase in the outlines of the pericardial sac, the diagnosis becomes extremely difficult. Under such circumstances the liberation by either aspiration of the serum or resection of a rib and thorough drainage of the pus is, we think, essential to an opinion. With the relief of the fluid the heart will return toward its normal site. If there remain an unusual area of precordial dulness there is a great probability that the pericardium is also filled. The great frequency of involvement of both the pleura and pericardium in pneumonic cases must be remembered. Of course, change in position of the patient will in large pleural effusion or empyema alter the upper line of dulness. This will, however, give you but little information with regard to the pericardium. Loculated empyema is usually latent and extremely difficult to localize. Unless large it is not apt to dislocate the heart except when present in the anterolateral part of the chest. When so situated the heart is dislocated to the right or left and a visible impulse and apex beat can still be

seen. The symptoms of a loculated empyema, cough, fever, and sweats, with the consequent leukocytosis persist. The pulse remains good, the heart sounds are readily heard and are not weak. There is no evidence of pericardial friction.

CASE I. (Reported in *New York and Philadelphia Medical Journal*, January 30, 1904.)—An Italian laborer admitted to hospital on the sixth day of pneumonia of the right base. Consolidation of left base on ninth day, with an extensive pleurisy. Pericarditis diagnosed on the thirteenth day. Paracentesis performed with immediate relief. Reaccumulation and formal operation on January 31st under local anæsthesia. Slow but complete convalescence at the end of six weeks' time.

M. D., an Italian laborer, aged thirty-six years (Hospital No. 3215), admitted to the Pennsylvania Hospital on January 4, 1903, with history of six days' illness, beginning with a chill, cough, and



CASE I.—The upper line shows the outline of dulness when the pericardium was filled with purulent fluid; the smaller outline of cardiac dulness six weeks after operation.

pain in the right side. On admission physical signs of consolidation of the right base were present. From the start the pulse was weak and heart sounds feeble, though the heart was not displaced and cardiac outline normal. Respirations were rapid (48 to 56), and the leukocytes (ninth day) 8800. On the ninth day the base of the left lung showed signs of consolidation with extensive pleurisy, which was especially marked in the left mammary region. The temperature for the first four days ranged between 103° and 102° F., and gradually fell by the twelfth day to 100° F. in the morning with an evening rise of a degree or more. This low temperature was maintained from this time on. The right base cleared slowly. By the thirteenth day the patient appeared very sick, with grayish

pallor, dyspnœa, and weak, rapid pulse. No pericardial friction was heard, but the pericardium was considered involved by the thirteenth day, empyema of the right base having been excluded by puncture. The apex beat now was neither visible nor palpable and there was impaired resonance in the fifth and fourth interspaces to the right of the sternum. This area of cardiac dulness increased, especially on the right side. The cardiac sounds were very indistinct. The epigastric region was full and very tender. The paradoxical pulse was marked on the eighteenth day of illness, January 16th. The leukocytes, before not increased, had now risen to 18,600. An exploring needle inserted in the fourth right intercostal space showed the presence of light-greenish, turbid fluid, which on culture showed a pure pneumococcic growth. The pericardium was aspirated at the same spot the following day and 420 c.c. of seropurulent fluid were withdrawn, with great relief to the more urgent symptoms. In a week's time rapid reaccumulation occurred, and, as was expected, exploratory puncture now showed thick, yellowish pus in the pericardium. The patient refused operation primarily. Permission to drain the pericardium having finally been secured, Dr. Le Conte operated on January 31st. Chloroform anæsthesia was at first attempted, but resulted in such struggling and cyanosis that local anæsthesia with cocaine was at once substituted and the fifth space incised at the site of the normal apex beat. The pericardium was readily opened and between a pint and a quart of non-odorous pus slowly escaped. The laboratory examination of the fluid showed many leukocytes and pneumococci.

The man was very ill for two weeks after operation, with weak, irregular pulse and but slight fever. From February 22d to 27th there was slight œdema of the face, back, and ankles present, though the urine during this time was free from casts, only leukocytes being present. At no time subsequent to the operation were pericardial friction sounds heard. The drainage tube was removed in twenty-six days and the patient was permitted to leave the hospital on February 23d, fifty-one days after operation.

It is interesting to note in this case the very mild degree of fever present while the pericardium was filled with seropurulent and purulent material.

This patient was seen December 31, 1903, almost a year after operation. He looks well and has gained flesh, and has been doing light work in a store. He states that he is sleepy a good deal of the time and has, on deep inspiration, some pain over the heart and in the back. No dyspnœa or cough is present. Appetite and digestion are good. Pulse is 76, small in volume, and very compressible. There is no œdema of feet. Liver is normal in size.

Inspection of Chest. No venous phenomena of neck; no retraction of interspaces over the cardiac area or behind (Broadbent's sign). The outline of the cardiac dulness is normal. The apex

in the erect position is in the fifth interspace within the midclavicular line. It is definitely movable with change in position. When he lies upon the right side the apex falls at least an inch toward the sternum, and lying on the left side pulsation is visible and palpable one inch outside of the nipple.

Auscultation. First sound is rather short, somewhat liquid, and with weak muscular tone. Second sound is clear; pulmonic sounds are distinct and normal, while the aortic sounds are rather weak and clear.

The features of interest in this case are:

1. The absence of leukocytosis during the pneumonia and its development with the pericarditis.
2. The absence of the pericardial friction sounds, both before and after operation.
3. The absence of fever, subnormal temperature with pus in pericardium.
4. Recovery without the physical signs of an adhesive pericarditis.

CASE II.—This case is one of lobar pneumonia complicated with empyema resulting in operation, with death eight days after operation; pyopericarditis overlooked.

The man, S. J. B., aged fifty-two years, was admitted to the Pennsylvania Hospital on December 21, 1903 (No. 2860). His family history is unimportant. Except for a pleurisy four years ago, which lasted three months, has had no serious illness. No venereal history. Formerly drank excessively, but periodically. Smokes freely. Is an attendant for feeble-minded children in Vineland.

Eight days before admission to the hospital he had a severe chill, which was followed by pain in the right side. He had a slight cough with little whitish expectoration. On admission he presented unusually small but equal pupils, urinous breath, with dry, parched tongue. Both the apex and base of the right lung are consolidated; the middle lobe escapes. The liver is increased in size. The abdomen is large; distention marked. The heart is either dislocated or increased in size, the right border being the right edge of the sternum, the left border anterior axillary line, the upper border the second interspace. The sounds are heard distinctly, but are not strong. The apex beat is palpable two inches outside the midclavicular line in the fifth space. No murmurs are present. The temperature on admission and for the first forty-eight hours was about 102° F. On the ninth day there was a fall to 100° F., with subsequent rise (pseudocrisis), and on the eleventh day to 99° F. From this time the temperature is but slightly elevated, about 100° F. The most extreme tympanites developed, which, it is interesting to note, was relieved by eserine. By the twentieth day of the sickness, or twelve days after admission, the signs at the right base indicated fluid. At this time, however, the leukocytes had fallen from 22,700 on the fourteenth day to 12,600. Exploratory puncture on February

5th showed the presence of pus. At this time it was noted that there was impaired resonance in the third and fourth interspaces to the right of the sternum. It was, however, supposed to be due to the line of fluid. The fifth rib in midaxillary line was resected by Dr. Le Conte and showed the presence of a large quantity of thick, yellowish pus in the right pleural sac. He did not do well after the operation, the pulse growing weaker and dyspnœa increasing; the temperature remained low. I regret to say that I did not see him after he was operated upon.

The autopsy showed, in addition to an unresolved pneumonia of the upper and portions of the middle and lower lobe, that the right pleural cavity was well drained, but that a purulent exudate filling the pericardium had been entirely overlooked. The pericardium contained 600 c.c. of thick, yellowish pus.

Case III.—Croupous pneumonia of the left base with the development of pericardial friction on the sixth day of illness; partial disappearance of the friction, with disappearance of the apex beat and great increase in the area of cardiac dulness. No evidence of fluid at the base at this time; incision into the pericardium, evacuation of pus; second operation in eleven days after the first, with drainage of some pus from the left pleura; rapid convalescence and recovery.

D. S. (No. 3141), aged thirty-three years, an Italian laborer, admitted to the Pennsylvania Hospital on January 14, 1904, complaining of pain in the left chest and back, with cough and dyspnœa, and fever of twenty-four hours' duration. On the second day it was evident he had consolidation of the lung involving the left base, faint bronchial breathing being heard at the extreme base and in the anterior axillary line. From the start the pulse was rapid, weak, and compressible. The man was very ill with active delirium. Complained much of pain, especially in the epigastric region and around the left costal margin. On admission the heart dulness was normal in size, no friction sounds were heard, and the heart sounds at the apex were very weak. On January 18th, or the sixth day of the illness, pericardial friction sounds were heard in the left third, fourth, and fifth intercostal spaces. The apex was not visible but palpable in the fourth interspace. By this time bronchial breathing was readily heard all over the left base. On the ninth and tenth days friction sounds were heard at the apex and to the right of the sternum; by the twelfth day there was dulness in the interspaces to the right of the sternum, with very weak pulse, dulness, and absent tactile fremitus in the left axilla. There was no evidence of fluid posteriorly. The leukocytes on the third day of the illness were 17,900; on the seventh day, 16,400; on the tenth day, or the period during which pericardial friction and increase in the area of cardiac dulness occurred, 28,000.

The temperature ran up to 103° F. until the seventh day, when a

pseudocrisis occurred with a fall to 100° F., subsequent rise and fall, and on the ninth day to 99° F. The pericardium was considered filled on the twelfth day. An exploring needle was introduced in the fifth interspace one inch within midclavicular line, and purulent fluid readily secured. Operation was performed within one-half hour under Schleich solution by Dr. Le Conte.

About a quart of pus welled through the incision in the fifth interspace. Upon sudden paroxysmal coughing a large quantity of pus, estimated two quarts, was suddenly expelled from the chest through the incision. It seemed as though an empyema must have ruptured through the incision. It is estimated that three quarts of pus were removed, which showed on culture pure pneumococcus growth. For eleven days the temperature remained between 100° F. in the morning and 102° F. in the evening, with rather rapid pulse (112 to 116); respirations about 32.

Unfortunately, the wound was permitted to close up about six or seven days after this operation. The case about this time was transferred to Dr. Harte's care, and after consultation with him it was considered advisable to reopen the chest to establish drainage, this time into the pleura, with the result that a small quantity of pus was recovered. This was done on February 6th and the twenty-fifth day of the illness. From this time on his convalescence was rapid and complete.

This case has been the subject of considerable discussion among his physicians; it has been questioned if the primary operation opened the pericardial sac, and the opinion expressed by those who did not see the case medically was that we are here dealing with an empyema and not pyopericarditis; that is, a loculated empyema was present with dislocation of the heart to the right side, and that the operation opened up the pleural sac and not the pericardium.

Case IV. (Seen in consultation with Dr. Richard H. Harte in the surgical wards of the Pennsylvania Hospital.)—N. M., an Italian laborer, aged twenty-two years. Both of his feet were frozen and he subsequently developed moist gangrene. Operation (amputation of both feet at lower third leg) after twenty-four days. Profoundly septic at this time. Sloughing of flaps, with granulation. Tardy convalescence. From the middle of February and through March the development of temperature with extensive increase in the precordial area of dulness; lungs clear. Operation, with drainage of the pericardium, on March 14th, with gradual convalescence.

N. M., aged twenty-two years, laborer, admitted to the Pennsylvania Hospital on December 21, 1903. During the cold weather of December this man slept in a barn, in which he was found on the day of admission with both feet frozen. On admission they were perfectly cold and of slate color to about four inches above the ankle. In twenty-four hours blebs formed over both heels, with return of normal color to the skin above the ankles. Moist

gangrene developed in several days' time. By January 10th the lines of demarcation present over both insteps with the development of septic temperature. He refused operation, but finally double amputation was consented to and done on January 14th. The flaps, however, did not do well, sloughed and healed slowly by granulation. By January 29th the temperature reached and remained normal for some time. During February, however, there was a gradual increase in temperature without any unfavorable signs. Pulse and respirations increased in frequency with sweats at intervals. No pain or cough or chill. It is noted, however, that the heart is dislocated to the right and visible pulsation is present in the third, fourth, and fifth interspaces, and the apex of the heart seems to be almost at the right nipple. On March 3d, examination shows that the cardiac dulness extends one inch to the right of sternum. The heart's action is embryonal. There is no cough or expectoration. On March 13th Dr. Harte asked me to see this case with him. There was present a large circular area of dulness over the præcordia, extending one and a half to two inches to the right of the sternum, to the second rib and outside the left midclavicular line. No visible pulsation is present. The heart sounds are loudest to the right of sternum. No friction is present. Paradoxical pulse not present. No venous phenomena in neck. The base and axillary region of left lung are clear. A needle inserted into the fifth interspace within the midclavicular line secures light-yellow, purulent fluid, which on examination shows the presence of streptococci. The following day Dr. Harte resected the fourth and fifth ribs within the nipple and secured a large quantity of pus. The man has slowly convalesced, but the heart has not returned to its normal position. There is still visible pulsation in the third right space, while the apex seems to be in fourth space (adherent pericardium?). No Broadbent sign. The points of interest in this case are:

1. The hæmatogenous or embolic origin of the pericarditis.
2. The unusual delimitation of the pericardial outline.
3. The absence of cough and pain.
4. The differential diagnosis between purulent pericarditis and dislocation of the heart by loculated empyema.

SURGICAL CONSIDERATIONS, BY ROBERT G. LE CONTE, M.D.

In presenting the surgical side of pyothorax the limited time at our disposal will prevent a discussion of the subject in full, and we therefore propose to deal only with three questions of importance to the surgeon: 1st, exploratory puncture for diagnostic purposes; 2d, anæsthesia; 3d, intercostal incision or excision of costal cartilages.

1. *Exploratory Puncture.* Purulent pericarditis cannot be definitely diagnosed unless the fluid is in hand for inspection. For this

reason exploratory puncture is an essential procedure in making the diagnosis. As we cannot tell the presence of pus by the degree of severity of the symptoms, the feeble pulse, and cardiac action, etc., therefore a formal operation for opening the pericardium should not be undertaken until pus has been proved present. One drop is sufficient for a diagnosis, and this can usually be obtained with a slender needle similar to the ordinary hypodermic needle, but somewhat longer.

The three principal dangers in exploratory puncture are wounding the heart, opening a large bloodvessel, and infecting the pleura if septic material be encountered in the pericardium.

A wound of the heart is grave according to its size and depth, but a small wound need not of necessity cause any symptoms. The cavity of the ventricles has been aspirated and life saved in acute dilatation of the heart, and it is a recognized and legitimate surgical procedure. If, therefore, a small needle is used no serious complication need result if the heart is punctured. When the heart is adherent to the anterior surface of the pericardium, or when the diagnosis of pericardial distention is at fault, there is great probability that the heart muscle will be penetrated by the needle. Should the needle puncture the heart it would readily be recognized by the motion imparted to it.

For the same reason the dangers resulting from the wounding of a large bloodvessel are insignificant, and need not be regarded if our needle is small. We can readily avoid the usual position of the internal mammary vessels, and no other vessels need be feared except in the presence of some anomalous distribution.

To appreciate properly the dangers of invading the pleural cavity during paracentesis we must turn for a moment to the anatomy of the part. The anterior mediastinal space, equivalent to that portion of the pericardium which is not covered by the pleuræ, practically commences at the level of the upper border of the fourth costal cartilage and extends downward to the diaphragm. The left pleura recedes much farther from the median line than the right, so that usually the edges of the left pleura do not reach the border of the sternum. For the purpose of obtaining some definite knowledge on the width of the anterior mediastinal space, 11 observations were made on the post-mortem table, and we are indebted to Dr. W. T. Longcope, the pathologist of the Pennsylvania Hospital, for the careful measurements and notes of these cases. From these observations we judge that the average width of the anterior mediastinal space, on a level with the fifth costal cartilage, is from 4 to 5 cm.; but that the space is quite variable is shown by the measurements, in one case being 8 cm. and in another 1 cm. In the latter case, with a width of 1 cm., the edge of the left pleura just reached the border of the sternum. In two other cases, where the widths were 3 and 4 cm., respectively, the left pleura reached nearly to the

edge of the sternum. In the remaining cases the border of the pleura was some distance from the sternum. The right pleura in every instance reached beneath the sternum.

In this region the left pleura is rather firmly attached to the triangularis sterni muscle, but can easily be dissected from it. Its attachment to the pericardium is a little more firm, but in a normal subject it can readily be separated until it dips over the side of the pericardial sac. For some distance, then, dependent upon the position assumed by the pericardium in the left chest, these two layers of parietal pleura are in close apposition. The substance of the membrane is so thin, frail, and translucent that it would be hard to conceive of any inflammation which involved the pericardium that would not also extend to both layers of the overlying pleura and obliterate the pleural space in this region.

The pericardium, although it does not contain elastic fibres, is capable of very great distention, and its cavity will hold quite two litres (Osler). The lungs are displaced and may be thrust far backward and outward in the presence of such distention, but the pleuræ, on account of their attachments, keep their usual relations to the anterior thoracic wall. That pathological lesions change the pleural relations of the pericardium very little is shown by the dissections of Delorme and Mignon (*Revue de chirurg.*, December, 1895, p. 830) in cases of pericarditis and in bodies where fluid was injected into the pericardium. We cannot compare distention of the bladder and its concomitant displacement of the parietal peritoneum with distention of the pericardium, for the peritoneum in the region of the bladder is very loosely attached to the anterior abdominal wall, while the pleural attachment is much more firm to the triangularis sterni muscle and still more firm to the pericardium. We therefore agree with Roberts in believing that Fowler is in error when he states that the boundaries of the pleuræ are greatly displaced in pericardial distention. As the pericardium probably distends equally in all directions the anterior mediastinal space must be broadened to some degree by this portion of the pericardium distending proportionately with the remainder of the walls of the sac. The more the pericardium is distended the greater will be the surface of pleural contact, and when this surface adheres the larger will be the operative field where the pleura can be crossed with safety.

To return to the observations on the cadaver, pins were thrust in the fourth and fifth interspaces to the right and left of the sternum, hugging the border of the sternum as closely as possible. It was found that on the left side the pins traversed the cavity of the pleura in both the fourth and fifth interspaces in one subject, where the breadth of the anterior mediastinal space was 1 cm.; that in two other subjects, in which the anterior mediastinal space was respectively 3 and 4 cm. broad, the pins in the fourth and fifth left interspaces grazed the pleura, but did not penetrate its cavity. In

the other eight cases the pins entered the pericardium some distance from the pleural border. On the right side, in every instance, the pins passed through the pleura before reaching the pericardium.

With such anatomical data we conclude that the point of election for paracentesis, or exploratory puncture, is in the fourth or fifth left interspace, as close to the edge of the sternum as the needle can be made to enter. In this position there is the smallest chance of penetrating the pleura, and the internal mammary vessels should lie well to the outer side of the needle. If exploratory puncture is practised in the fifth right interspace, the point suggested by Rotch, the pleura will almost invariably be traversed. Puncture in the space to the left of the xiphoid cartilage necessitates passing through the diaphragm, and therefore dangerously close to the peritoneum.

The ordinary hypodermic syringe, armed with a needle an inch and a quarter or an inch and a half long, is introduced in the fifth left interspace. If no fluid is withdrawn, and there is no evidence of penetration of the heart by motion imparted to the needle, the fourth interspace should be tried. If this also fails to demonstrate fluid, the same interspace one and one-half or two inches from the left border of the sternum should be tried. If septic material is not found, there can be no danger of infecting the pleura, even though its cavity is invaded. If septic material is present in the pericardium the overlying pleural space is almost certain to be obliterated, and under such circumstances the needle can pass through it without danger of further infection.

2. *Anæsthesia.* In the presence of a large collection of fluid, with feeble heart sounds, the administration of a general anæsthetic becomes very dangerous. In Case I. the patient after four or five inspirations of chloroform became very cyanosed, with feeble, fluttering pulse, and death seemed imminent. Under such circumstances we are forced to use a local anæsthetic, and our experience with infiltrations of Schleich's fluid has proved most satisfactory. In such cases where the heart action has not been seriously interfered with, ether would seem to be the best for general anæsthesia.

3. *Intercostal Incision and Excision of Costal Cartilages.* The choice of operation will be very greatly influenced by the condition of the patient. If a general anæsthetic is contraindicated, excision of a costal cartilage will be dangerous, for under local anæsthesia it cannot be done without causing considerable pain, and severe pain would be very depressing to the heart. Under such circumstances our choice will be limited to an incision through an intercostal space, or aspiration of a large part of the fluid for the purpose of temporarily relieving the embarrassed heart and permitting later the giving of a general anæsthetic.

Incision is certainly as safe as aspiration with a large needle or trocar, and it frequently permits of a recovery without further operative procedure, while aspiration necessitates a later operation.

If the pericardium is much distended with pus the overlying pleural cavity is almost certain to be obliterated, and incision through it can be made without risk.

The point of election for incision is the fourth left interspace, beginning one inch from the sternal border and extending to the normal position of the apex beat of the heart, about one inch internal to the anterior mammary line. The tissues are infiltrated with Schleich's fluid and the incision gradually deepened. When the pleura is adherent it cannot be recognized; but if its cavity is free, and the incision has been carefully deepened, air will rush in as soon as it is penetrated, and before the pericardium has been opened. When pus is reached it is allowed to flow out slowly for fear of embarrassing the heart by the sudden relief of pressure. A rubber drainage tube is inserted and an ample dressing of gauze applied. This is sufficient for the time being, and the question of flushing the pericardium or resecting the costal cartilages may be put off until the physical signs show that the drainage is not good or that the tube is plugged with membranous lymph.

Intercostal incision produces no shock; it is the simplest procedure in grave cases; it permits of a more extensive operation at a later period if found inefficient; it permits the recognition of non-adherent pleural surfaces before the pericardium is opened, and in the majority of cases it will lead to a cure where a recovery is possible from an operative procedure. The question of the recovery of the patient is more dependent upon the cause of the pyothorax and the pathological lesions present in other portions of the body than upon the choice of an operative procedure.

The deductions drawn by Reichard ("Zur Kasuistik der Operationen bei Pericarditis," *Mitteilungen aus den Grenzgebeiten der Medizin und Chirurgie*, No. 7, p. 356), in advocating resection of a rib instead of incision in the intercostal space, do not seem to us to be justified. He collected 33 cases, in 19 of which incision was practised and in 14 resection of a costal cartilage. Of the 19 cases 6 recovered and 13 died, and of the 14 resections 8 recovered and 6 died. From this statement of figures he concludes that the safest procedure is resection. If, however, we examine these cases more carefully we will see that of the 13 cases dying after incision 1 died on the table, 2 others within twenty-four hours, a fourth while the pericardial cavity was being flushed after operation, a fifth some weeks later, following a double empyema operation, and a sixth from joint empyema. These 6 cases would probably just as surely have died after resection, and one is led to believe that perhaps their condition was so desperate that so formal an operation as resection was not used because too prolonged and dangerous. If, then, these 6 failures are subtracted from the 13 deaths, we would have 6 recoveries and 7 deaths following incision. Also, when we examine the cases that recovered under excision, we find

that in 1 the disease was primary in the pericardium, and that 2 others were the result of stab wounds—in a sense, therefore, primary also. Such favorable cases would probably have recovered as rapidly with simple incision, and, if so, by removing them from the tables of the recoveries we would have 5 recoveries with 6 deaths. In other words, the statistics of the two procedures may be regarded as very nearly equal in the same class of cases.

If excision becomes necessary we are inclined to think that the chondroplastic flap suggested by Roberts is better than the removal of one or two costal cartilages. Roberts cuts his flap through the fourth and fifth costal cartilages and turns it upward, using the soft tissues of the third interspace as a hinge. On the normal cadaver it gives a good exposure of the internal mammary vessels and readily permits of their ligation if they cross the field of operation, and the border of the left pleura is easily recognized and can readily be dissected free and pushed aside if its surfaces are not adherent.

CONCLUSIONS. 1. Purulent pericarditis is quite frequently overlooked in lobar pneumonia and in other pyæmic states.

2. Its presence modifies the pre-existing disease to a considerable extent.

3. In diseases with high temperature the presence of purulent pericarditis depresses the fever range and increases the respiratory and pulse rate without sufficient clinical evidence of trouble elsewhere than in the pericardium.

4. It is yet to be proved that the heart, in case effusion is present in the pericardium, is always dislocated backward and upward. It is probable that in some cases the presence of fluid in the pericardium dislocates the heart either to the right or to the left. (This, of course, will occur if previous adhesion between the heart and the pericardium has occurred on either the right or the left side.)

5. The diagnosis once made, the only treatment should be incision and thorough drainage. (Paracentesis will at times relieve temporary urgent symptoms and should be performed without the slightest fear.)

6. That exploratory puncture can be made with safety, and is essential for diagnostic purpose before operation is undertaken.

7. That such puncture should be made in the fourth or fifth left intercostal space, as close to the sternum as possible, with a fine needle.

8. That pericardial dilatation does not alter the relations of the pleuræ to the anterior thoracic wall.

9. In the presence of pyopericarditis the overlying pleural space is usually obliterated by extension of the inflammation.

10. That incision under local anæsthesia is the preferable operation in the majority of cases.

11. That Roberts' chondroplastic flap is preferable to an ordinary excision of costal cartilages when incision fails to give adequate drainage.

PERICARDITIS: ITS SYMPTOMS AND DIAGNOSIS.[1]

By David Riesman, M.D.,

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Pericarditis is, as a rule, more damaging to the heart than endocarditis. It is almost certain to shorten the life of the person afflicted unless an intercurrent disease should anticipate its effects.

Its causes, to which, merely for the sake of completeness, I would briefly refer, are in the main twofold: direct extension and hæmatogenous infection. Traumatic pericarditis is so rare that it may be disregarded.

The pericardium may be involved by direct extension from neighboring structures in the thorax, such as the lung, the pleura, the mediastinal and bronchial lymph glands, and the œsophagus; also from the endocardium or the myocardium, from the great vessels near their origin, and from aneurysms. Inflammatory processes below the diaphragm may likewise cause pericarditis by extension.

Hæmatogenous infection is the most frequent, and gives rise to the type of pericarditis accompanying acute articular rheumatism and other infectious diseases. A form of particular interest is that associated with Bright's disease and uræmia, the true nature of which is still in doubt. Unquestionably, however, its cause is hæmatogenous, and is either a toxin or a bacterium. It may develop in any of the forms of nephritis, but is most frequently encountered in acute Bright's disease, in the early stages of the large white kidney, and in chronic interstitial nephritis. Frederick T. Roberts says that it is almost never found in cases of typical large white kidney. In one of my cases, however, the kidneys were in an advanced stage of chronic parenchymatous nephritis (the so-called large white kidney); but, as the patient had pneumonia also, the share of the kidneys in producing the pericarditic process is not readily fixed.

Nephritic, or uræmic, pericarditis is nearly always of the fibrinous variety, with little fluid; but it may be serous or hemorrhagic. I am not forgetting simple hydropericardium, which is sometimes present in cases of chronic nephritis complicated with general dropsy and effusions into the serous cavities. I have, however, been surprised at the rarity with which hydropericardium occurs when the conditions supposed to favor it, renal and cardiac dropsy, are present.

[1] Read before the College of Physicians of Philadelphia, May 4, 1904.

Pericarditis is quite common in cases of pneumonia, especially when this disease is on the left side. It is frequently overlooked, but should always be thought of as one of the possible causes of so-called delayed resolution, or, rather, delayed crisis. In my experience it has usually been purulent; but it may be serofibrinous. It is generally accompanied with a mediastinopericarditis or a pleuropericarditis of the same nature.

I must say a few words in regard to pericarditis of tuberculous origin. This is usually of the plastic, obliterating variety, but sometimes it is purulent. It is rare, and is commonly overlooked during life; and even after death its true nature may not be recognizable without a microscopic examination.

In taking up now the symptoms and diagnosis I shall not discuss these two aspects of the subject separately, but shall consider the principal features as they present themselves to the examiner.

1. The Friction Sound. This is most marked in acute fibrinous pericarditis, the commonest form. Its site of maximum intensity is to the left of the sternum in the third and fourth interspaces, and sometimes in the second. In rare instances, as in two under my observation at the present time, it is heard first or solely at the apex. It is usually of a churning character, superficial, and markedly intensified by pressure with the stethoscope. It may have a gallop-rhythm, and in pronounced cases it has a peculiar chugging quality, like the noise made by a locomotive just starting to move. If confined to the apex, it may closely resemble a presystolic murmur; while when loudest at the base it may simulate double aortic disease. The character of the pulse, the size of the heart, and the history, together with careful examination so as to discover the second aortic sound, will aid in the diagnosis from aortic-valve disease.

When the friction sound is loud it is often difficult to distinguish existing endocardial murmurs. These may be heard if a double stethoscope is used and no pressure is applied. Pressure brings out the friction sound and thereby renders the murmurs faint or inaudible. I have recently been able to study the heart before the development of pericarditis in two cases. One was that of a young Italian with aortic regurgitation. In the other case, also that of an Italian, there was mitral insufficiency. As the friction sound increased in loudness, the murmurs in both grew fainter and fainter, and at present the aortic murmur is inaudible; while the mitral systolic can be heard when the stethoscope is placed lightly upon the chest. In some cases a mitral murmur, obscured in the precordial area by the confused noise of the friction sound, may be heard in the axilla.

Pleural and pleuropericardial friction must also be differentiated from the ordinary pericardial friction. Both are modified by respiration.

The existence of a friction sound does not exclude the presence of fluid. In one of the cases in which Dr. Roberts tapped and obtained considerable fluid, the friction sound had not disappeared. The fluid is often held in compartments separated from one another by bands or areas of adhesion, which preserve the friction sound. In some cases the friction sound persists at the apex after it has disappeared elsewhere, probably because the greater movability of the apex retards the formation of adhesions.

Friction may cause a palpable thrill. Of my recently observed cases this thrill was present in two out of nine. The loudest friction may, however, be entirely unaccompanied with thrill.

2. Pain and Tenderness. These were present in four of the nine cases. In these four the pain was intense. In three the tenderness was an exquisite hyperæsthesia, so that percussion became torture. In one of the cases the pain was paroxysmal, and was apparently as severe as in angina pectoris, although its direction of radiation was different. It started at the wrists and ran with great violence up the arm and across the shoulders into the chest. In another case there was moderate pain and tenderness in the epigastrium, and in one severe subjective pain in the præcordia and the epigastrium, without any tenderness.

It is not easy to explain why, with no difference in the physical signs, the pain and tenderness should be extreme in some cases and entirely absent in others. It may be that in those with pain there is involvement of the superficial structures, especially the muscles, analogous to that described by Coplin as accompanying pleurisy and pneumonia.

3. The Apex Beat. In the fibrinous form the apex impulse is diffuse, indistinct, and sometimes undulatory, and can usually be appreciated better by touch than by sight. In effusions of any size, it is commonly absent; but a more or less vigorous apex beat is entirely compatible with the presence of a large effusion, as was strikingly shown in a patient that came under my observation at the Polyclinic Hospital. Even when the apex beat cannot be seen, it may often be felt as a distinct shock—a point of importance in differentiating between dilatation and effusion. When the apex beat can be located it is found above the lower level of dulness.

4. Pericardial Effusion. Few conditions are more frequently overlooked than effusion into the pericardial sac. At times the signs are unmistakable, even upon very superficial examination; at other times strong doubt encompasses the diagnosis. The signs to which value should be attached are:

1. The character of the right border of the heart, which becomes convex.

2. A change in the cardiohepatic angle, which, from having been a right angle, becomes an obtuse one.

3. As a corollary, the presence of Rotch's sign, dulness in the right fifth interspace, close to the sternum.

4. An increase in dulness upward as far as the second interspace.

5. Flatness with intense resistance on percussion.

6. The position of the apex beat with relation to the lower boundary of the precordial dulness.

7. The shape of the precordial dulness, which is triangular, or, rather, shaped like a trapezium, with the base downward.

8. Bamberger's sign, a patch of dulness with bronchial breathing near the angle of the left scapula.

9. Ewart's sign, a separation of the first rib from the clavicle, making it possible to feel the former throughout its length.

10. The presence of fluid on exploratory puncture.

The best place for *exploratory puncture* and also for aspiration is still unsettled. There is certainly no one point of election suitable for all cases. When the presence of fluid is suspected and the friction sound is still present over limited areas, it is a good and self-evident rule not to make the puncture where friction is heard; nor should it be made at a point where the heart sounds are very distinct. Another wise maxim is to go in slowly and to arrest the needle as soon as the heart is felt to rub against its point. If the apex can be shown by auscultation—or perhaps by palpation—to be above the level of the fluid it will naturally be perfectly safe to tap below it. This will usually bring the point of puncture in the sixth interspace, about the nipple line. At other times the fourth or fifth interspace on the right may with advantage be selected.

The presence of fluid may be simulated by dilatation, and differentiation of the two conditions is far more difficult than the parallel-column method of text-books indicates. I have mistakenly tapped in a case of this kind, and others have had a similar experience. The position of the apex beat with relation to the lower boundary of the heart is of value in the diagnosis; also the presence of a decided cardiac impulse, especially a pronounced diastolic shock. Search should also be made for Bamberger's sign, referred to above.

Great diagnostic difficulties must arise when the fluid occupies an abnormal position; when, for instance, it is collected on the left side of the heart, in which case the precordial dulness extends into the left axilla and simulates pleural effusion. If the fluid is in the pericardial sac, the dulness rarely extends below the eighth rib in the axillary line. Sometimes only tapping can decide the question. When a pleural effusion is co-existent—and this is not at all uncommon—the discovery of the pericardial effusion is rendered far more difficult. When there is double pleural effusion, the pericardial effusion is usually overlooked.[1]

[1] In one of Thayer's cases, just published (Johns Hopkins Hospital Bulletin, May, 1904), the fluid (over 1200 c.c.) was lying posteriorly to the heart and at the lateral angles of the pericardial sac.

Sometimes pericardial effusions shift with change of posture, especially when the patient is turned to the right. Fluctuation is said to occur at times, but in the cases that I have seen it was not noticed.[1]

Pericardial effusion sometimes simulates *mediastinal tumor*. This is especially the case when the effusion is of long standing. In both conditions there may be dyspnœa, orthopnœa, engorgement of the jugular veins, intense cyanosis, dysphagia, enlargement of the veins of the chest, and extreme flatness on percussion. As at times there is no history suggesting an antecedent pericarditis, suspicion of a mediastinal tumor in these circumstances becomes not unreasonable. A year or two ago I reported a case of this character to the Section in Medicine of the College of Physicians. The patient had the signs that I have mentioned. The cyanosis and dyspnœa were very marked, and the flatness on percussion was most striking. The onset had been insidious, and there was no history of any previous illness. The man was not bedfast, but was able to walk to the Polyclinic Hospital on the clinic days. I took the view that the case was one of either pericardial effusion or mediastinal tumor, although the latter seemed the more likely; but the man died a few weeks later of exhaustion, and the autopsy showed hæmopericardium, the origin of which was not revealed. It is a curious fact that in another case in which the diagnosis of mediastinal growth was for a time entertained, hæmopericardium was also found.

Pericarditis with effusion sometimes simulates *pneumonia*. The distended sac compresses the left lung, and examination of the latter may show impaired resonance and a patch of bronchial breathing in the back. The existence of fever, cough, leukocytosis, dyspnœa, and cyanosis strengthens the suspicion of pneumonia. In a case of this kind that came under my observation at the Philadelphia Hospital, the cyanosis and general prostration were out of proportion to the extent of lung apparently involved. Such disproportion may constitute a point of diagnostic significance.

It is well known that morbid conditions in the chest may cause *pain to be referred to the abdomen*, and give rise to the belief that the disease is located below the diaphragm. I need mention only the so-called "abdominal" pneumonia, which is not rare in children and is also seen in adults. This may resemble appendicitis or perforation of the intestine. Less well known is the fact that pericarditis may cause pain of such severity in the abdomen as to simulate peritonitis.

On the other hand, the interesting observations of Ortner (*Wien. klin. Woch.*, June 29, 1899) teach us that in some cases of peritonitis, chiefly of the hepatic and epigastric regions, a friction sound

[1] In pericardial effusion, just as in hypertrophy of the heart, skodaic tympany may sometimes be detected over the upper part of the left lung.

may be heard over the præcordia, in the total absence of inflammation of the pericardial sac.

A peculiar case, probably belonging to the class described by Ortner, came under my observation at the Polyclinic Hospital in February. A woman, fifty-two years of age, was admitted with severe epigastric pain, tenderness, vomiting, slight jaundice, fever, and leukocytosis. There were no cardiac symptoms, but examination showed the presence of a pronounced friction sound, which was audible from February 3d to February 6th. A few days later a friction sound was discovered in the region of the gall-bladder, extending thence for a short distance upward over the liver. Within a few days this also had disappeared, and the patient left the hospital apparently well.

The only way in which one can escape the error of diagnosing abdominal disease when the trouble is in the chest, and *vice versa*, is by making a careful examination of the thoracic and abdominal organs in all cases.

Even in the early stages of pericarditis signs of *pulmonary congestion* are common, especially on the left side, in the lower axillary region. In three of my cases a distinct generalized *bronchitis* was found.

Fever is, as a rule, present in acute pericarditis, but it has nothing distinctive. When the effusion is purulent the fever may be intermittent, even when the quantity of pus is small. The fever in pericarditis may be present for several days before the friction sound is discovered. Until then the case is usually considered to be one of tuberculosis, sepsis, or typhoid fever. I have thought that in such circumstances the pericarditis probably begins posteriorly, and that the sudden appearance of the friction sound, several days after the onset of fever, is due merely to the fact that the inflammation has extended sufficiently far forward for the friction to become audible.

A curious phenomenon not rarely observed in pericarditis is pronounced *delirium* or other mental disturbance. Da Costa, Balfour, and Musser have made reference to this feature. In one case studied by me with the late Dr. Packard at the Philadelphia Hospital, there was maniacal delirium, which might readily have led an observer less thorough than Dr. Packard astray. Da Costa, in one of his last papers, advised that in every case of acute mania the heart should be examined for pericarditis. The duration of the delirium is variable. In a case of mine, one of tuberculous pericarditis, it extended over a number of weeks and was of a mild character. In his lucid intervals the patient was extremely irritable.

It is difficult to explain the variability in the general reaction of the system to inflammation of the serous membranes. We are accustomed to consider the three serous sacs identical; but pathology proves them to be different, even if histology does not.

The pleural cavity may contain pus for days, weeks, or months without the production of a serious general infection. The peritoneum is a much less efficient guardian of the organism, and suppurative inflammations, as a rule, very speedily influence the condition of the system at large. Acute inflammations of the pericardial sac, whether purulent or serofibrinous, seem to be rapidly productive of constitutional symptoms.

In order to solve these enigmas, we must know:

1. The conditions of lymphatic absorption, both as to direction and as to rapidity.

2. The efficiency of the lymph-gland outposts.

3. The richness in nerve supply, upon which the degree of functional disturbance will depend.

Adherent pericardium more often, probably, than any other important condition comes to the autopsy table undiagnosed. One reason for this is that at times the characteristic signs are altogether wanting. At others, some or all of them are present but are overlooked. Briefly stated these signs are as follows:

1. Systolic retraction of the intercostal spaces.

2. Retraction of the lower portions of the chest in the lateral and posterior aspects—the so-called Broadbent's sign.

3. Diastolic shock or rebound.

4. Absent respiratory movement in the epigastric triangle. The last three are found especially in cases of mediastinopericarditis.

5. Pulsus paradoxus—the so-called Kussmaul's sign—the pulse becoming weaker during inspiration. This is not of much diagnostic value, except when accompanied by inspiratory swelling of the veins of the neck.

6. Diastolic collapse of the veins of the neck—Friedreich's sign.

7. Marked undulation of the heart beat.

8. Early ascites, preceding œdema of the legs.

9. Marked irritability of temper. I have been struck with this, but cannot say that it is either a constant or an important symptom.

10. In rare cases, mild delirium.

For practical purposes the following statements may be made with regard to adherent pericardium. It may be suspected:

1. In cases in which a friction sound has been heard over a great extent of the præcordia and has disappeared.

2. As a probability, in children having a chronic valvular lesion and marked hypertrophy of the heart. In such cases adherent pericardium is commonly found at autopsy. The extreme stretching of the sac and the consequent pressure of the two layers against each other cause alterations of the endothelium favoring a low-grade infection.

3. In adults and children with chronic valvular disease, in whom, with heart sounds of good volume and an impulse of great force, there develop ascites and dropsy of the limbs. In such cases we

may infer that there is something more than the mere valvular lesion. The ancillary factor may not always be adherent pericardium; it may be a fibroid myocarditis, but it is often the former.

4. In the cases of so-called quiet ascites, chiefly occurring in young subjects, in which cirrhosis of the liver and tuberculous peritonitis can be excluded. I have reference to the so-called pericarditic pseudocirrhosis, a subject ably discussed before this society by Dr. Kelly.

Since the beginning of the year to the present time, 9 cases of acute pericarditis have come under my observation: 2 I saw through the courtesy of my colleague, Dr. Thomas G. Ashton, at the Philadelphia Hospital; 2 occurred in my own wards in that institution; 3 were observed during my term of service at the Polyclinic Hospital; 1 was in a patient of Dr. Francis T. Stewart, and 1 I saw in consultation with Dr. L. Podolski. Of these 9 cases 3 were in the female and 6 in the male sex; 4 of the patients were colored persons; 3 were tapped—1 with a negative result.

Etiologically the cases may be classified as follows: Probably of rheumatic origin, 4; associated with pneumonia and nephritis, 1; secondary to operation for the radical cure of hernia, 1; secondary to gallstone colic, 1; of unknown origin, 2. Of the 9 cases, 7 were associated with endocarditis and 2 were unaccompanied with any evidence of endocardial lesion; 2 ended fatally, the autopsy showing fibrinous pericarditis with adhesions.

I have confined myself to a mention of these few details in regard to the cases so as not to lengthen the paper unduly. Those interested in the subject will find these cases more fully discussed in the forthcoming volume of the *Transactions of the College of Physicians of Philadelphia*, 1904, vol. xxvi.

TWO CASES OF PARACENTESIS OF THE PERICARDIUM.[1]

By John B. Roberts, M.D.,

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The glory of modern surgery can be appreciated only by practitioners who, in their youth, were familiar with the almost universal occurrence of operative sepsis. Surgical timidity, due to "inflammatory fever" and its results, made the experienced surgeons of our youthful days skeptical of the opinions of those of us who saw the coming of the new era.

The contention that trephining was not a serious procedure and ought to be adopted in nearly all fractures of the cranial vault[2]

[1] Read before the College of Physicians of Philadelphia, May 4, 1904.

[2] Roberts, Operative Surgery of Human Brain, Transactions of the American Surgical Association, 1885.

aroused opposition, and the advocacy of suprapubic cystotomy in preference to perineal incision was regarded as foolishness.[1]

So it was with my proposition that the diseased pericardium should be treated as the pleura in similar pathological conditions. That the pericardium should be promptly aspirated (1876), if it contained a serous or hemorrhagic effusion unaffected by the administration of drugs, and that it be kept empty by incision and drainage (1876), if the fluid contents were purulent,[2] was doubted. These procedures are now routine operations. It then seemed hazardous to incise the chest wall and pericardium to clear up a doubtful diagnosis of dilated heart or pericardial effusion (1881).[3] The advocacy of heart suture as a therapeutic procedure for wounds of the cardiac wall (1881 and 1883) seemed in 1882 to be a youthful indiscretion, if one could judge from the tone of the Fellows of this College.[4] A formal method of attacking the pericardium and heart by a trap-door resection of the costal cartilages, devised in 1897,[5] was, however, the outcome of these studies.

This obstructive conservatism is, however, the usual attitude of mind of the middle-aged, and surgeons of that period of life need to bear in mind that most of what the profession learns is taught by young men.

To-night several cases of pericardicentesis and of pericardial drainage have been reported, and two Fellows, Dr. John H. Gibbon and Dr. Francis T. Stewart, have each added to surgical literature an instance of heart suture for penetrating wound. In Dr. Stewart's case the patient recovered. I feel honored that both are ex-resident physicians of the Polyclinic Hospital.

Through the courtesy of Dr. David Riesman I recently had the opportunity of aspirating the pericardium in two patients with pericarditis, and desire to join him in the record of the cases. Both were tapped with the pericardial aspirating trocar devised and described in 1877 and 1881.

Roberts' pericardial aspirating trocar, consisting of a hollow needle and an internal cannula with flexible end.

Case I.—The patient, a colored woman, had been admitted to the Polyclinic Hospital the day before with pericarditis and bronchopneumonia.

Tapping was done March 9, 1904, at 6 P.M., in the fourth left

[1] Roberts, Transactions of the American Surgical Association, 1884, p. 169.
[2] New York Medical Journal, December, 1876.
[3] Annals of Anatomy and Surgery, December, 1881, p. 250; Medical News, January 13, 1883.
[4] Transactions of the College of Physicians of Philadelphia, 1883.
[5] The American Journal of the Medical Sciences, December, 1897.

interspace over the apex beat, which could be distinctly felt. The point of puncture was about three and one-half inches to the left of the middle line of the sternum, under the left mammary gland, and perhaps through its lower edge. The Roberts pericardial trocar was employed, and worked satisfactorily, the vacuum of the aspirating pump being attached after a little fluid had flowed from the trocar. The introduction of the needle, which was made without attaching the aspirator, was followed by pushing forward the internal cannula. The trocar was introduced to a depth of about 3½ cm. The point gave the impression of being within a cavity, and the beat of the heart against it was easily felt. The external portion of the trocar oscillated with the cardiac pulsations. Three ounces of a very bloody serum were drawn off. That this was not pure blood was shown by the lightish color and the yellowish-red stain made on the towels when some drops soiled them. In order to make a strong vacuum, after the small bottle was about half-filled, the aspirating apparatus was detached. The contents of the bottle had coagulated so promptly that it took a little time to wash out the bottle and to obtain some of the fluid in sterile test-tubes for bacteriological examination. The aspirator was attached to the trocar again, but no further fluid was obtained. When the aspirating apparatus was detached there was a rush of air into the bottle, showing that a vacuum had been properly obtained. No air flowed into the chest, as the trocar on being withdrawn was found to be plugged with a clot of bloody lymph.

The wound was sealed with collodion and cotton. I thought it not wise to withdraw the inner cannula to remove any possible plug, because this would have left the sharp point of the needle exposed and liable to lacerate the heart wall.

Percussion over the upper cardiac region after the patient had been tapped showed clearness where previously there had been dulness. The woman, after the operation, seemed to breathe more easily, and had a better pulse. She was somewhat excited during the operation, but did not seem to suffer much real pain. Ethyl chloride was used to freeze the skin before the puncture was made.

Before the operation there was dulness of the right chest posteriorly, with moist rales. There was marked dulness over the heart. The dull area was somewhat irregularly triangular, with the base downward. This dulness extended into the left axillary region. The dull area seemed to be displaced forward when the patient turned on the right side. The cardiac sounds and impulse were quite distinct in front, and a friction sound was heard at the base of the heart. This was said to be less than on the previous day. The cardiac sounds were heard posteriorly. There were some rales posteriorly in the left chest. The patient was breathing very rapidly, and had a weak, frequent pulse, but was able to lie flat on

her back even before operation. The day after operation she was in better condition than on the day of operation, and the cardiac dulness, which had been diminished by operation, was not increased.

On March 12, 1904, at 12.30 P.M., it was determined to make another aspiration, because the pericardial sac had evidently refilled. A puncture was made with the Roberts trocar in the fifth interspace, two and three-quarter inches to the left of the middle line of the sternum. The aspirator pump was attached, but nothing was obtained, except a very few drops of bloody fluid. The trocar was then withdrawn and found to be plugged. A second puncture was then made in the same interspace, three and three-quarter inches from the mid-line of the sternum. The puncture, made a few days previously, had been made, in the interspace above, three and a half inches from the mid-line of the sternum and through the lower portion of the breast gland. The second puncture, made to-day, obtained by aspiration four and one-half fluidounces of dark-bloody fluid. The fluid was much darker than that obtained a few days ago, and did not coagulate so quickly as that did, if it coagulated at all. It was thicker than the previously obtained fluid. The trocar moved with the heart beats in both punctures to-day, but not so vigorously as it did on the former occasion. At the last puncture the trocar was buried in the tissues about 9 cm. The hand of the operator received the impression that the point was in a large cavity. At the first puncture the end seemed not to enter a large cavity, but to strike against some solid body within the chest. An increased resonance was obtained by percussion of the precordial region after the operation. The patient died early the next(?) morning.

The autopsy showed the pericardial cavity nearly free from fluid; the heart was covered with shaggy lymph, particularly about its base, and there were adhesions between the heart and pericardium. The punctures in the chest wall were in the fourth and fifth interspaces, and the first puncture made on the second day scraped or went through the edge of the costal cartilage above the space.

Upon the front of the heart was a small ecchymosis without apparent wound that looked as if it might have been caused by contact with the end of the trocar. This fact could not be definitely confirmed, as the point of ecchymosis was not very marked. There was a considerable amount of fluid in the left pleural cavity, and a condition of pleuropneumonia. There was some congestion and consolidation of the right lung.

CASE II.—Colored man, aged thirty-one years, in the Polyclinic Hospital under Dr. Riesman. Admitted on March 22, 1904; died on March 28, 1904.

On March 24, 1904, at 11 P.M., I found an enlarged area of cardiac dulness, and valvular murmurs present and easily heard. The

apex beat was seen and felt under the left nipple. I did not believe there was much fluid in the pericardium, though the dulness extended to the right of the sternum. Heart was supposed to be enlarged. Some dulness of the right chest existed posteriorly, with some harsh breathing. At the right base posteriorly there was heard what seemed like pleural friction, but I was uncertain whether it was not rales. Left chest posteriorly showed some dulness. The question in my mind was whether the pulmonary congestion was secondary to the heart lesion, or whether the old cardiac valvular disease had the pulmonary condition added to it and was causing weak pulse (130?), rapid respiration, and sweating, and dyspnœa. The man was not sitting up to breathe. I decided that there was not enough fluid in the pericardium to justify tapping. I had not the benefit of Dr. Riesman's presence to aid me in the diagnosis.

Dr. Riesman sent word on March 25th that he thought tapping was desirable, but could not stay to the operation. I saw the patient and found the condition to be about as the day before, but more dulness in the cardiac area appeared to be present. I tapped in the fourth right interspace close to the sternum with Roberts' aspirating trocar, not attached to vacuum until after insertion. Three fluidrachms of slightly reddish fluid were obtained. The trocar did not move with the heart, but with respiration.

I then punctured in the fifth left interspace directly over the apex beat about one and one-half inches below the nipple, as it seemed desirable to be sure there was no large effusion overlooked. The end of the trocar seemed to strike the heart and perhaps to be embedded in the heart muscle, because it moved with such vigor with the heart beats. No fluid, except a few drops of blood, escaped. The pump brought very little fluid, and, after repeated attempts to get fluid, the instrument was withdrawn. Attempts were made to obtain fluid by altering the position of the end of the cannula, but none was obtained. The end did not feel to be free in a cavity of any size. The end of the cannula contained blood clot, and there was a free flow of blood from the wound until it was closed with cotton and collodion. The patient at one time complained of severe pain, and it was then that the operator felt that the heart had been wounded.

The next day the patient seemed better; the pulse was stronger, and it was thought that the area of cardiac dulness on the right of the sternum was less.

Patient died on March 28th. Autopsy showed no pleural effusion; no fluid in pericardium, but marked amount of lymph in pericardium. The heart was greatly hypertrophied. No mark was found of wound of heart, but this would probably have been concealed by lymph deposits.

CANCER OF THE PANCREAS AND GLYCOSURIA.

AN HISTOLOGICAL STUDY OF THE CHANGES IN THE ISLANDS OF LANGERHANS.

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This investigation was undertaken for the purpose of determining what relation, if any, the islands of Langerhans may have to the glycosuria occasionally seen in connection with cancer of the pancreas. That pathological changes in these structures are frequently associated with diabetes has been satisfactorily demonstrated by the investigations of Opie[1] and others, and has led to a more definite knowledge of a form of diabetes which may properly be called pancreatic diabetes. No attempt has been made on the other hand, as far as I am aware, to demonstrate the presence of similar changes in connection with the glycosuria occasionally associated with cancer of the pancreas. That some of these changes —hyaline degeneration, chronic interacinar pancreatitis involving the islands, and, according to a few investigators, diminution in the number of the islands—might result from the involvement of the organ by cancer is evident from our knowledge of the secondary lesions frequently accompanying such invasion. It is well known that duct obstruction, frequently the result of cancer of the head of the pancreas, readily causes a chronic pancreatitis; and it is possible also that the destruction of a large portion of the pancreas parenchyma by the new-growth may destroy the islands to such an extent that they are no longer equal to the demands made upon them.

On the other hand we have no adequate explanation of those cases in which, though the pancreas is extensively or completely replaced by the new-growth, diabetes does not exist. Such complete destruction, as in the cases reported by Litten[2] and v. Hansemann,[3] is analogous to the extirpation experiments by v. Mering and Minkowski,[4] but the absence of glycosuria is opposed to their results. v. Hansemann has attempted to explain the discrepancy by assuming that in primary cancer of the pancreas the tumor cells originating from pancreas parenchyma inherit the function of internal secretion peculiar to the normal cell. This explanation, however, is entirely hypothetical, and in the light of our present knowledge of pancreatic diabetes loses its force unless we assume that all primary cancers of the pancreas arise from the islands of Langerhans, which, despite the recent publication of Fabozzi[5] supporting such origin appears most improbable. If our newer theories concerning the relation of the islands to carbohydrate metabolism

are firmly grounded we would expect to find evidence of the persistence of the islands in such cases. With these various possibilities in mind, I have made a careful histological study of 30 cases of cancer of the pancreas, in 3 of which glycosuria was present.

The literature offers but scanty information concerning the frequency with which glycosuria occurs in connection with cancer of the pancreas. Such cases are not uncommonly reported, but without reference to the frequency of cases not complicated by glycosuria. From a hasty review of the subject I believe the proportion (1 in 10) shown by my cases to be high. We have, however, definite information concerning the reverse condition; that is, the frequency of cancer of the pancreas in connection with all forms of diabetes. In Oser's[6] series of 188 cases of diabetes, cancer occurred in 24; in Williamson's[7] series of 23, once; and in 139 cases reported by Windle,[8] in 3. v. Hansemann's[9] tabulation of 72 cases of diabetes with pancreatic lesion contains 5 cases of cancer. I have examined many of the original descriptions of the cases quoted in these series, with the object of determining if any peculiar macroscopic or microscopic conditions existed. The task has been most unsatisfactory, for many of the reports are of a clinical nature and gross and histological descriptions are meagre or entirely lacking. This analysis, however, confirms the well-known clinical fact that in the majority of cases the new-growth is in the head of the pancreas and by duct obstruction causes an atrophy (chronic pancreatitis?) of the distal portion of the organ. This point is especially emphasized by v. Hansemann, Dieckhoff,[10] and Windle.

The material upon which this study is based consists of the tissues from 30 cases of cancer selected from a group of 35 examples of new-growth in the pancreas. As the material has been gathered from different sources, often from routine collections of autopsy material, it has not always been possible to procure tissues from the different portions of the uninvolved pancreas as well as from the cancerous portion, nor for the same reason have essential data concerning clinical history and pathology been available in all cases.* Thus while all cases have been utilized in the study of the histological changes but 21 have been available, on account of incomplete examination of the urine, in arriving at conclusions concerning the occurrence of glycosuria. The 30 cancers studied may be classified as follows: primary in the head of the pancreas, 9; in other portions of the organ, 4; secondary to gall-bladder or ducts, 5; to stomach, 8; to breast, 1; and to uterus, 1. Glycosuria occurred in but 3 cases, in 2 of which the tumor was primary in

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the head of the pancreas; concerning the third, no clinical or pathological notes were available.

The results of the histological examination, especially as concerns the islands of Langerhans, are more readily described under the following headings: changes in (*a*) the immediate neighborhood of the new-growth, (*b*) at a distance from the tumor, (*c*) in cases with extensive involvement, but no glycosuria, and (*d*) in cases accompanied by glycosuria.

(*a*) The important changes at the edge of the advancing cancer are the persistence and increase in size of the islands of Langerhans. While the glandular elements are invaded and replaced by the new-growth and the newly formed connective tissue, the islands remain unaltered in the midst of the stroma and occasionally are even enclosed by a mass of tumor cells. Not only is this true of the edge of the tumor, but in well-infiltrated parts, the islands may be seen in the stroma without remnants of acini. The increase in size of some of these structures is four to five times that of the normal islands. These giant islands are typical in structure and readily distinguished from small alveoli of cancer cells. They represent a true hypertrophy of the islands and not altered pancreas acini.

(*b*) The changes at a distance from the tumor depend apparently upon the location and size of the tumor. With a cancer of the head of the pancreas causing pressure upon the duct a chronic interstitial pancreatitis of severe grade always existed, which, with the exception of one case to be described later, was always of the interlobular type. The islands persist in the midst of dense connective tissue and are not invaded by it. This condition has been observed by Opie in several similar cases without glycosuria. A tumor at the head of the pancreas not obstructing the duct does not cause chronic interstitial changes. Small tumors of any portion of the pancreas are unaccompanied by changes in the islands of the normal portion of the organ; on the other hand, if the new-growth is of considerable size, definite hypertrophy of these structures is apparent.

(*c*) But two cases of diffuse involvement of the organ occurred in this series; in neither was glycosuria present. These are, however, not analogous to those discussed by Hansemann, for areas of normal pancreas could be found here and there, lying in the midst of the new-growth. In such areas were large islands several times the normal size.

(*d*) Three cases of cancer of the pancreas accompanied by glycosuria are included in this series. One of these could not be utilized in the present study because of lack of data; the gross description of the pancreas not being available and the histological study incomplete, inasmuch as only the tumor had been preserved and a study of the uninvolved portion of the pancreas was therefore impossible. The second case, a primary cancer of the pancreas involving the head only and accompanied by a slight intermittent glycosuria

with free fat in the stools, showed no changes in the uninvolved portion of the organ. The islands were few in number and the increase in size usually seen in the immediate vicinity of the new-growth was not evident. The third case was one of cancer of the pancreas with advanced chronic pancreatitis of the interacinar type. Sugar had been present in the urine for several months before death. The entire length of the pancreas was 15 cm.; the proximal 5 cm. occupied by the tumor, the remainder of the organ atrophied and indurated. Histological examination revealed a severe grade of chronic pancreatitis with small, scattered groups of more or less altered acini. Within these, smaller strands of connective tissue had penetrated, involving to a varying extent the islands of Langerhans.

While it is impossible to draw any definite conclusions from such a small number of cases these observations are very suggestive and offer some support of the newer ideas concerning the anatomy and physiology of the islands. The persistence of the islands in the midst of the tumor stroma supports the theory of anatomical independence; an independence which I have recently definitely demonstrated by a study of their development.[11] This persistence is analogous to that described by Schlesinger,[12] Opie, Condon,[13] and Pearce[14] in congenital syphilitic pancreatitis, and by Opie in various other sclerotic conditions of the organ. The increase in size of the islands suggests a purposeful enlargement due to increased function, and therefore of the nature of a compensatory hypertrophy. A similar hypertrophy of these structures has been described by Warthin[15] and by Ohlmacher[16] in connection with cancer of the liver, and is considered by them also to be compensatory in nature, compensating, however, for the lost or limited liver function. The livers of my cases have not been available in all instances for histological examination, and I have not therefore been able to arrive at any conclusion concerning the relation of enlarged islands to lesions of that organ. It is possible that a relation of a compensatory nature may exist between the carbohydrate metabolism of the liver and the islands of Langerhans; but the hypertrophy of the latter structures when associated with cancer of the pancreas is, I believe, an attempt to compensate for the function of the lost islands.

The cases of diffuse cancer of the pancreas without glycosuria can be explained only on the supposition that the amount of normal pancreas remaining is sufficient to furnish the secretion necessary to carbohydrate metabolism. v. Mering and Minkowski found in their extirpation experiments that if one-fourth to one-fifth of the pancreas remained, diabetes did not result; a destruction of the pancreas by cancer we may consider analogous to partial extirpation. In both of my cases of diffuse cancer small areas of pancreas containing islands could be found in the midst of the new-

growth in a sufficient amount to correspond to the unextirpated fifth. In this connection the possibility of an accessory pancreas (Thorel,[17] Sweet[18]) or other glands maintaining the balance of function must also be considered. v. Hansemann's explanation of the absence of glycosuria is not applicable unless we assume that all cancers of the pancreas arise from the cells of the islands of Langerhans.

Finally, we have the cases with glycosuria. In one of these a true diabetes existed; in the other an intermittent glycosuria. The first is readily explained by the presence of an interacinar pancreatitis with involvement of the islands, a type of pancreatitis originally described by Opie in connection with diabetes and since observed by many investigators. It therefore appears that while cancer of the head of the organ with duct obstruction may frequently cause a chronic pancreatitis, glycosuria does not result unless the interacinar form with involvement of the islands develops. This naturally brings cancer diabetes into a common relation with pancreatic diabetes in general.

The case of intermittent glycosuria brings up the vexed question of alimentary glycosuria. It is well known that a normal individual may digest without disturbance from 150 to 250 grams of sugar, but that an increase over this amount may lead to temporary excretion of glucose in the urine. This excretion due to hyperglycæmia is the inevitable result of a sudden flooding of the circulation with an amount of glucose beyond that which the body is able at once to consume. On the other hand, in a number of abnormal conditions where there is no actual diabetes, an alimentary glycosuria may be produced by an amount of sugar which in the economy of the normal individual is easily consumed. Among the prominent conditions in which this occurs may be mentioned neuroses, traumatic or otherwise, organic nervous diseases, alcoholism, infectious diseases, exophthalmic goitre, hepatic and pancreatic disease. Is it not possible that this temporary excretion of sugar is due to some transient disturbance of the function of the islands brought about by vascular disturbances of the islands or by some toxic substance, perhaps of the nature of an autotoxin, affecting the secretion of these structures or temporarily throwing them out of function? In a destructive disease of the pancreas, as cancer, a certain number of the islands are necessarily destroyed; the remainder, however, may be sufficient under normal circumstances to furnish the necessary secretion, whatever it may be, essential to carbohydrate metabolism; but if overtaxed by an unusual injection of glucose, hyperglycæmia and a consequent glycosuria might result. This explanation has occurred to me in connection with my case of cancer accompanied by intermittent glycosuria; and that it is not entirely hypothetical is indicated by the observations of Ssobolew,[19] confirmed by Herzog,[20] that in some cases of permanent diabetes the

pancreas exhibits a great diminution in the number of islands and in some cases an entire disappearance. This deficiency, however, is far from proven, and in mentioning it I do not wish to be placed in the position of advocating a pancreatic origin for all forms of diabetes.

CONCLUSIONS.—We have a definite basis for ascribing a certain portion of the cases of diabetes to lesions of the pancreas and ultimately to lesions of the islands of Langerhans. As a result of the study here briefly outlined we can, I believe, include the permanent diabetes of cancer of the pancreas in this group. Further observation must be made before the intermittent glycosuria can be attributed to destruction of large numbers of islands. The absence of diabetes in cases of extensive involvement of the pancreas would appear to be due to the survival of small fragments of pancreas, including a number of islands sufficient to carry on their peculiar function. The persistence of the islands under the adverse conditions described supports the observations heretofore made concerning their anatomical independence, while their enlargement, suggesting as it does a compensatory hypertrophy, is corroborative of the theory that they have an independent special function.

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ASCARIDES IN THE BILE-DUCTS SIMULATING GALLSTONE SEIZURES.

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Intestinal worms are not considered common causes of biliary colic; they are, perhaps, not sufficiently considered in this role, for when we remember that the natural habitat of these parasites is the small intestine, and that not infrequently do they pass into the common duct and there effectually occlude the lumen of that canal and completely obstruct the flow of bile, it is obvious that their presence would give rise, quite naturally, to the complex chain of symptoms noted in the occlusion of that duct from other causes.

Where the natural current of normal bile is not interfered with it is a sterile fluid. This has been proven experimentally on dogs by Netter[1] and confirmed by Gilbert and Girade,[2] and further confirmed by Naunyn,[3] who reports a case wherein the bile was sterile two hours after death.

Bile has also been regarded an antiseptic, and to this property has been ascribed the function of preventing decomposition during digestion; but this premise has failed to receive sufficient proof, while on the other hand it has been proven that it does not possess any antiseptic properties. In the report of a case of biliary fistula Mayo-Robson observed that the absence of bile from the intestine for a period of fifteen months did not lead to any irregular fermentation processes, and concludes "that the alleged antiseptic effect of bile on the feces is probably imaginary."[4] Professor Birch has reached the same conclusion[5] after numerous cultivation experiments.

So much, then, in consideration of normal bile when its natural current is not impeded.

In emphatic contrast to the researches noted above we know, from equally able investigators, that when the flow of bile along the ducts is for any reason arrested, micro-organisms *usually do* invade the gall-bladder, on the hypothesis that they gain entrance either from the blood or from the intestines.

Charcot and Gombault[6] were the first to demonstrate this fact by ligating the common duct in dogs; later this was confirmed by Netter,[7] who, after an aseptic ligature of the common duct in dogs, found that both staphylococcus and bacillus coli commune could be cultivated from the stagnant bile. Bouchard[8] cultivated the bacillus coli commune from the bile of cases suffering with disease of the biliary tract which was of sufficient severity to produce inflammatory changes on the mucous lining of the ducts, thus oblit-

erating the lumen of the canal, and in consequence producing a stagnation of the bile.

In answer to the query, "How do these bacteria reach the gall-bladder?" Mayo-Robson[9] gives three possible sources, viz.: (*a*) through the bile-ducts, or (*b*) from the blood, or (*c*) directly through the wall of the gall-bladder.

It is, then, only in diseased conditions that these micro-organisms are present in bile, and it seems that the biliary apparatus will tolerate a moderate degree of inflammatory involvement and be perfectly able to maintain its integrity, providing the flow of bile is not interfered with, but cannot withstand the closure of the chief duct and resist the invasion of the bacteria.

This fact, then, becomes the pivotal point which must determine the causes of gall-bladder infections. It makes no difference whether the bile current is stagnated by an inflammatory process involving the duct to the degree of occluding its patency, or whether there is an impacted gallstone in the common duct, or whether an ascaris has wandered from its habitat and entered the common duct, or the simple aseptic ligature, experimentally performed; the result is the same, the flow of normal bile is prohibited, and bacterial invasion is certain to be a consequence.

It is not surprising, then, that the case cited below was admitted to the hospital and the opinion given of probable impacted gall-stone in the common duct:

A. S., aged thirty-four years, Austro-Hungarian. Engaged in railroad grade work. Lives in a temporary camp with other men of his class. Surroundings very unhygienic. Food improperly prepared and of poor quality.

April 12, 1904. *Present History.* Complains of pain in stomach, no appetite, tongue coated and foul, fetid breath. Has had attacks of severe colic, lasting a few hours, leaving a sense of soreness in epigastrium. Temperature, 99° F.; pulse, 80; anxious facial expression.

19*th.* Admitted to Mercy Hospital. Deeply jaundiced; mucous membranes of the mouth and conjunctiva yellow; sclerotic membranes of both eyes deeply jaundiced. Hepatic area of dulness much increased, border of liver extending 6 cm. below costal arch; exceedingly tender on slight pressure over site of gall-bladder, tumor evident between the costal margin of ninth rib and umbilicus. Pulse, 60° F.; temperature, 98.4; respiration 16. Frontal headache, anorexia, and eructations of gas. Constipated. Urine: dark-brown colored; specific gravity, 1028; reaction, acid; chlorides, 16 per cent.; phosphates, 8 per cent.; sulphates, 8 per cent.; albumin, none; sugar, none. Biliary pigments distinct by Ulzmann's test. Biliary acids were detected by Dr. Oliver's test,[10] and estimated by his standard table as follows: To 4 c.c. of Oliver's solution 1.25 c.c. of urine was added; a very distinct milkiness appeared at once.

In the second trial 4 c.c. of the test solution was added to 1 c.c. of urine, with prompt milkiness. In the third trial 4 c.c. of the test solution was added to 0.60 c.c. of urine, resulting in no immediate reaction, but in a short time slight milkiness occurred. According to Oliver's table of standards this would indicate a percentage increase of 600. Stool almost white, and about the consistency of putty.

Patient was ordered a liquid diet, and, as was suggested by Mayo-Robson,[11] he was given calcii chloridi, 2 grams three times daily, to lessen the tendency to hemorrhage during the operation. Calomel 0.65 every hour for three doses, follow at once with $MgSO_4$, 15 grams in half-glass of hot water.

20th. Pulse, 49; temperature, 97° F.; respirations, 16. No bowel movements. Urinated about 300 c.c. during the night, highly colored. Pain quite severe in stomach. Deep jaundice; headache. Calomel 0.65 every hour for three doses, follow at once with $MgSO_4$, 15 grams in hot water. Calcii chloridi, 2 grams three times daily.

21st. Pulse, 58; temperature, 97.8° F.; respirations, 16. Bowels have not moved; urinated four times. Complains of headache. Deeply jaundiced. Pain constant. Calomel reordered as above, followed by $MgSO_4$. Calcii chloridi continued. Prepared for operation.

23d. Pulse, 52; temperature, 98.2° F.; respirations, 16. No breakfast. Had four bowel movements, clay colored, thin and offensive; the second movement occurred at 5 A.M., and was allowed to remain in the bed-pan and placed in the closet. The nurse reported a long, brown body in the evacuation, and Dr. J. M. Crowley, the assistant superintendent of the hospital, examined the evacuation and secured a live ascaris lumbricoides 28 cm. long. On my arrival at the hospital, Dr. Crowley and I examined the evacuation for the ovæ, but none were found. It was thought that possibly this worm was responsible for the symptoms manifested, and operation was postponed. Santonini, 0.65, with calomel, 1.95, was ordered at noon, to be repeated at 9 P.M., and followed by Ol. ricini 30 c.c. in the morning.

24th. Slept well. Bowels moved four times freely, clay colored, thin; urine dark. Deeply jaundiced.

The subsequent history is not of especial interest. In a few days the pain subsided, the liver area of dulness became normal, no pain on pressure could be elicited, tongue cleaned, appetite returned, jaundice gradually faded, and he was discharged May 5, 1904.

It might be of interest to note that, while the patient began to improve in every way and the appetite was fully restored, while the bowels moved regularly every day, it was not until the day previous to his discharge from the hospital that the feces resumed their natural color, and when the coloring of the bile did appear in

the stools it was not gradual but spontaneous; the evacuation preceding the one of natural color was as cream colored as they were when he first entered the hospital.

Mertens[12] has reported forty-eight cases of ascarides in the bile-ducts, but in only five cases were the symptoms such as would suggest the presence of gallstones, and in but two of these was the cause determined during life.

In his own case, a woman of thirty years of age was admitted to a hospital following the third attack of biliary colic, which began with pain in the gall-bladder region, associated with vomiting, and followed by deep jaundice; both liver and spleen were increased in size. The patient rapidly became emaciated for seven weeks, at the end of which time she passed two partially macerated round worms, and she rapidly improved and ultimately recovered.

When we consider that these worms inhabit the upper portion of the small intestine, and recall the migratory nature of the parasite, the wonder is not that they occasionally wander into the common duct, but that they are not found there more frequently than observers would lead us to believe.

Their presence in the bile-duct certainly interferes with the normal flow of the bile and invites bacterial invasion by stagnation of that fluid, and I believe that in the cases cited above such an infection occurred, and this premise is substantiated by the tardy appearance of the bile in the stools. There is no doubt but that the gall-bladder and the bile-ducts were infected and suffered inflammatory hypertrophy sufficient to completely obliterate the lumen of the common duct, and even after the removal of the provoking cause were slow to repair this damage and functionate normally. The pain symptom further emphasizes the truth of this statement.

Kehr[13] states that pain in gallstone disease is not due, as is commonly supposed, to the pressure of a hard substance held in a soft mucous-membrane tract, either in the cyst or the ducts; the mere presence of stone in these passages may interfere with the normal currents of the bile, and that bacterial invasion which follows this impeded current invade the gall-bladder and its ducts. The result of this inflammation is the softening of the mucous membrane of the gall-bladder and the ducts, and the transudation of serous or purulent fluid into these hollow organs; at first the transudation takes place by reason of the increased circulation attracted to this site by the bacterial invasion of the coats of either the gall-bladder or the ducts, and when first poured out is a serous fluid; later, as the bacterial invasion increases, this serous fluid is readily converted into a purulent fluid.

In either case the gall-bladder and its appendages are distended, and the nerve endings throughout its surface are put upon the strætch and dragged upon, and this gives the symptoms of pain, and is solely excited by the distention of the walls of the gall-bladder and ducts.

It is not the character of the fluid in those hollow organs that determines the pain, but the fact that *any* fluid has produced distention sufficient to violate the nerve endings.

Kehr[14] mentions a simple interesting experiment in proof of this statement: following an operation on the gall-bladder where there is a fistula established, inject into the gall-bladder physiological salt solution; the moment distention occurs, complaint will be made by the patient of severe cramp in the stomach.

The jaundice of the case was very pronounced; all visible tissues were deeply stained; the skin was dry and harsh. This color appeared quite abruptly, following shortly after my first examination on April 12th, and was of the same deep color that was so characteristic a feature until the passage of the worm on April 23d; after this the color gradually faded.

It seems that these round worms are very commonly found in autopsies upon laborers of this class, especially among the Italian. Dr. F. F. Agnew, formerly of Blockley Hospital, Philadelphia, stated to me in a conversation recently that in a number of autopsies during his service he cannot recall an instance when these worms were not found in this class of laborers. Thus it would seem that the mode of living and the sanitary surroundings had a determining influence.

The hospital record shows that this patient complained bitterly of pain, and was not able to sleep well from the date of his admission, April 19th, until the date on which he passed the worm, April 23d. After that date his appetite improved and he slept well, and when seen he would make deep pressure over the former site of his pain, which was a little to the right of the epigastric median line, and say in English, "No pain," and shake his head vigorously to emphasize the statement.

There were no special symptoms that might lead one to suspect the presence of ascarides; the clinical picture was purely one of biliary colic, and, combined with the quickly developing jaundice and the continued pain, presented a typical case of obstruction of bile from whatever cause.

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ACUTE PRIMARY CHOLECYSTITIS, WITH A REPORT OF FOUR CASES.

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Potain,[1] in 1882, and Courvoisier,[2] in 1890, described acute cholecystitis as a rare but rapidly fatal disease. In 1898 Richardson[3] published an article that should have convinced every practitioner that acute inflammation of the gall-bladder occurs not only as a complication of gallstones, or secondary to other diseases, but also as a primary and serious affection characterized by signs and symptoms that correspond to the extent and intensity of the pathological changes. I believe that most medical men are rather doubtful as to the occurrence of acute primary cholecystitis, and many still believe in such statements as the following: "Acute empyema, or gangrene of the gall-bladder, is associated with gallstones in 74 per cent. of the cases. In the remainder it occurs in zymotic diseases, as typhus fever, variola,"[4] etc. The time has come when we should discard such statements. We should know that acute primary cholecystitis does occur, that it can be diagnosticated, and that in most cases it can be treated successfully.

In this paper I shall discuss only cases of acute inflammation of the gall-bladder in which gallstones were not found, and in which the condition could not be considered secondary to any other disease.

Case I. *Sudden onset; chill; biliary colic; history of two primary attacks; operation; cholecystitis found; many adhesions; no gallstones; chronic appendicitis; recovery.*—Joseph B., a miner aged twenty-nine years, was seized with a violent chill. This occurred about midnight. Shortly afterward he began to suffer from violent paroxysmal pain in the abdomen. He vomited and the pain disappeared. In a few minutes the colic returned. The pain was very severe, was most intense in the pit of the stomach, and radiated to the right scapular region. Between midnight and 4 A.M. he had six paroxysms of pain. Examination (4 A.M., June 16, 1901) was as follows: The patient was in shock. Pulse rate, 110. There was no fever. His tongue was coated. The pupils were slightly dilated, but reacted normally. There was no jaundice. Examination of the heart and lungs was negative. The abdomen was slightly distended and was tender. The region of the gall-bladder was extremely tender. The right rectus was more tense than was the left. The remainder of the examination was negative. I washed out his stomach and gave him three-eighths of a grain of morphine and one-one-hundred-and-fiftieth of a grain of atropine hypodermically.

Previous History. The patient had measles when a child. He had never suffered from typhoid, variola, or pneumonia. Two years previously he was ill for one week with an attack similar to the present sickness. During this illness the pains were very severe, and he thought he had fever. Eight months ago he suffered from an illness that confined him to his bed for two weeks. The pain at this time was severe, and was paroxysmal for five days. Then it became continuous and gradually subsided at the end of two weeks. The region of the gall-bladder was tender for four weeks following this illness. The patient had never been jaundiced.

Operation. The morphine gave very little relief. The paroxysms of pain continued. At 8 A.M. his temperature was 101°, and his pulse rate 110. The patient was etherized at 9 A.M. After anæsthesia was complete I was unable to feel the gall-bladder or to determine that the liver was enlarged. An incision was made along the outer border of the right rectus muscle, extending from the costal arch downward. The omentum, colon, and gall-bladder were firmly matted together. I freed the gall-bladder and found that it was slightly enlarged, was very tense, and of a very dark color. I then brought the appendix into the wound. It showed evidences of chronic inflammation. After removing the appendix the gall-bladder was surrounded with gauze, aspirated, sutured to the transversalis fascia, and opened. It contained very dark and thick bile. The mucosa of the gall-bladder was œdematous and injected. No gallstones were found.

The patient made an uneventful recovery. The drainage was profuse, but contained no stones. The temperature and pulse rate became normal on the fourth day. The fistula was closed at the end of six weeks, and the patient has been well since that time.

A smear from the gall-bladder contents showed the presence of a short bacillus that stained well with methylene blue.

CASE II. *Chill; colic; high temperature and pulse rate; local tenderness over the gall-bladder; operation; gall-bladder enlarged, tense, and showed beginning gangrene in one place; cholecystostomy; no stones found; recovery.*—George T., aged twenty-four years, was seized suddenly with severe abdominal pain. The pain was paroxysmal, was located in the epigastrium, and did not radiate. He vomited twice. He had a severe chill, but cannot remember when this occurred. He is of the opinion that the chill preceded the pain. I saw him, with Dr. B. B. Slick, of Ridgway, Colorado, twelve hours after the onset of his trouble. At this time his temperature was 104° and his pulse rate 146. The pain had ceased. The region of the gall-bladder was sensitive on deep pressure. The right rectus muscle was rigid. The gall-bladder could not be palpated. Dr. Slick told me that the paroxysms of pain were very severe for ten hours and that one-quarter of a grain of morphine

hypodermically had no effect on them. The patient's previous history showed that he had never been sick before.

Operation. Local anæsthesia was used. The gall-bladder was exposed through a vertical incision. It was enlarged and distended. The fundus showed an area of beginning gangrene. The gall-bladder was aspirated, sutured to the transversalis fascia, and opened. The organ was sufficiently enlarged so that it could be brought far enough into the wound to suture beyond the gangrenous area. The gall-bladder contained dark, thick material.

The patient made a rapid recovery.

No bacteriological examinations were made.

CASE III. *Chill; colic; fever; local tenderness; vomiting; operation; gall-bladder not perforated; general peritonitis; gangrenous cholecystitis; no gallstones; death.*—Mrs. S., aged thirty-six years, complained of violent abdominal pain about 11 P.M. At about the same time she had a violent chill. She vomited several times. The pain was paroxysmal, was located in the pit of the stomach, and did not radiate. She was given one-quarter of a grain of morphine hypodermically every hour. One and one-quarter grains were given before she was relieved. She then slept for six hours. When she awoke the pain returned, but was less severe. After the first thirty-six hours the paroxysms of pain ceased and she complained only of a dull, continuous pain. She ceased vomiting at the end of twenty-four hours, and did not begin to vomit again until the sixty-fourth hour. The bowels moved on the second day in response to five grains of calomel and an enema. The temperature ran from 101° to 103°; the pulse rate from 110 to 120. There was no jaundice. She had rheumatism fourteen years ago, and for eight years had suffered dyspnœa after exertion. For five years she had suffered more or less from the pain and hemorrhage caused by a uterine fibroid. She had had no acute infectious diseases.

I saw the patient seventy-two hours after the beginning of the illness. At this time the temperature was 104° and the pulse rate 130. The abdomen was distended, rigid, tympanitic, and tender. The region of the gall-bladder was more sensitive than the remainder of the abdomen. Examination of the lungs was negative. The heart was enlarged transversely. A systolic murmur was present over the apex and in the axilla. There was a leukocytosis of 22,000. The urine was scanty and contained much albumin. The liver dulness was slightly increased in size.

Operation. A longitudinal incision was made over the gall-bladder. The liver was enlarged. The gall-bladder was somewhat enlarged and distended. It was very dark in color, but was not gangrenous or perforated. There were no adhesions. General suppurative peritonitis was present. The appendix was normal. No tumor was felt in the region of the pancreas. The gall-bladder was aspirated and found to contain pus. A rubber tube

and strips of gauze were placed through the posterior abdominal wall, on the right side, to drain the peritoneal cavity. Then the gall-bladder was anchored and opened. No stones were found. The patient died from general peritonitis forty-eight hours after the operation was performed.

Autopsy. General suppurative peritonitis; acute cholecystitis and cholangitis; fibrous myocarditis, with fatty degeneration; parenchymatous degeneration of the spleen, liver, and kidneys; a moderate degree of chronic interstitial nephritis. Atheromatous areas were present in the aorta and in the coronary arteries. There was a fibroid involving the fundus of the uterus.

CASE IV. *Acute primary cholecystitis; gangrene of the gall-bladder, with perforation; general peritonitis; death; no gallstones found at operation or autopsy.*—Carl H., a brakeman, aged twenty-seven years, who had had no previous sickness, was suddenly taken ill with a chill and with violent paroxysmal pains in the abdomen. The pain radiated, at times, to the right scapular region. He vomited continuously. His temperature averaged 103° and his pulse rate 120. A small bowel movement was obtained about thirty-six hours after the onset of the disease. On the second day of his illness the pain became continuous. There was no jaundice. There was marked tenderness over the region of the gall-bladder. The abdomen was distended, tender, and rigid.

Operation, fifty-five hours after the beginning of the illness, showed a gangrenous and perforated gall-bladder, with general suppurative peritonitis. The appendix and pancreas showed no marked changes. The patient died thirty hours after the operation. No gallstones were found at operation or autopsy.

CASE V.—M. Richardson[3] reports a case of acute cholecystitis occurring in a man, aged forty years, whose only previous illness had been an attack of "inflammation of the stomach," five years previously. The patient complained of acute pain in the abdomen. Twelve hours afterward his temperature was 101°. The abdomen was rigid and was diffusely tender. These symptoms were most marked on the right side. Operation revealed acute cholecystitis. A cholecystostomy was done and the patient recovered. The colon bacillus was found in the gall-bladder.

CASE VI. (Reported by Richardson.[3])—A man, aged twenty-eight years, who had never been sick, complained of paroxysmal pains in the groins. A hypodermic of morphine did not give him relief. The temperature was 102° and the pulse rate 108. A laparotomy revealed a general peritonitis. The gall-bladder was gangrenous, but was not perforated. No gallstones were found. The patient died on the fourth day following the operation.

CASE VII. (Reported by Richardson.[3])—A man, aged fifty-six years, who had previously had some pain in the region of the gall-bladder, was suddenly taken ill with abdominal pain, vomiting,

fever. Temperature 103° and pulse rate 100. The area over the gall-bladder was markedly tender. In this case the patient had accurately located the point of greatest tenderness without advice or suggestions. Cholecystostomy revealed acute cholecystitis. The gall-bladder contained bacilli and cocci. No gallstones were found. The patient recovered.

CASE VIII.—W. A. Lane[5] reports a case of acute primary cholecystitis occurring in a man, aged fifty-four years, who had always been in perfect health. The patient was suddenly seized with severe paroxysmal abdominal pain, prostration, and constipation. Attempts to produce evacuation of the bowels were tried for three days, but were unsuccessful. Laparotomy showed the duodenum and a portion of the colon covered with a layer of lymph. The gall-bladder was much distended, but was not gangrenous or perforated. A cholecystostomy was done. The gall-bladder contained mucopus but no stones. The patient made a perfect recovery.

CASE IX.—E. M. Pond[6] reports a case of acute primary cholecystitis occurring in a healthy man aged sixty-five years. The onset was sudden, the pain paroxysmal, and the abdomen very rigid and tender. The area over the gall-bladder was extremely tender. Operation revealed a gangrenous and perforated gall-bladder; general peritonitis; no gallstones. The gangrenous portion of the gall-bladder was removed and drainage employed. The patient recovered.

CASE X.—A. J. Ochsner[7] reports a case of cholecystitis occurring in a woman aged fifty-two years. She was sick one week previous to being operated upon. During this time she had four attacks of severe pain in the epigastrium. The gall-bladder was found greatly distended with thick, dark bile. No gallstones were found. A cholecystostomy was done and the patient recovered.

CASE XI.—Curtis[8] reports a case of acute fatal peritonitis in which operation revealed advanced cholecystitis without perforation as the cause. The presence of gallstones was not mentioned.

OCCURRENCE. Acute primary cholecystitis is not such a rare condition as is generally supposed. Musser[9] states that the primary form of acute cholecystitis is more common than is usually thought. M. Richardson states that acute cholecystitis, without known pre-existing gall-bladder disease, is more common than intestinal obstruction. The reports of large numbers of cases of operations on the biliary apparatus show plainly that cholecystitis —acute, chronic, with and without gallstones—is not an uncommon disease. But these reports are of little value in determining the relative frequency of occurrence of the acute primary form. The experience of any man who does a large amount of surgery and little else does not give a fair estimate of the relative frequency of occurrence of diseases like acute primary cholecystitis, for the

following reasons: The large majority of the patients, coming to the surgeon of large experience, have first passed through the hands of the general practitioner. Many of these cases are chronic. But the rule is that, although the general practitioner may not have made a diagnosis, he is usually reasonably certain that the case is a surgical one before he refers it to the "surgical specialist." The chronic sufferers may find their own way to the surgeon of large experience, but in the great majority of cases it is the general practitioner who directs the plan of procedure in the acute serious diseases. For these reasons, the man who does very little outside of operative work is at a disadvantage in regard to diseases like acute primary cholecystitis. It is impossible, at the present time, to determine the frequency of occurrence of acute primary cholecystitis. I am of the opinion that acute and subacute primary cholecystitis is much more common than any of us would believe.

ETIOLOGY. I am unable to make any positive statements in regard to the etiology. It is probable that the colon bacillus is most frequently the cause of the inflammation. It is possible that the typhoid bacillus may remain latent in the gall-bladder for a number of years and then cause an acute attack of cholecystitis. Hunner[15] reports such a case. These patients usually give a history of a previous attack of typhoid fever. However, Cushing[11] reports one case of gallstones, with acute typhoid cholecystitis, in which there was no history of a previous attack of typhoid.

It has not been positively determined how the micro-organisms reach the gall-bladder. It is generally believed that they come from the intestinal tract. The usual idea has been that they reached the gall-bladder through the common and cystic ducts. Cushing[12] states that in cholecystitis following typhoid the organisms get into the gall-bladder by way of the portal vein. I have thought that the mechanism of infection might be as follows:

When the colon bacillus finds its way into the portal vein it seldom passes beyond the liver. Many times it is excreted in the bile. Now some of these organisms, in being excreted from the liver, might be carried by the bile into the gall-bladder. Some of the organisms might lodge between the mucous folds of the cystic duct and excite an inflammation. The swelling of the lining of the duct would soon close the outlet of the gall-bladder. This would lower the resistance of the gall-bladder mucosa (Charco and Gombault[13] and Netter[14]), and, according to Leubuscher, do away with the antiseptic properties of the bile in the gall-bladder. (Cushing says that the bile is not only not antiseptic, but is a good culture medium.) If we accept the foregoing as the process by which the inflammation starts, we can easily see how distention, gangrene, and perforation of the gall-bladder, and peritonitis, with or without perforation, might occur, according to the virulence of the infection and the resistance of the patient.

PATHOLOGY. I can add nothing to the pathology that has not been given in the report of the cases. It would seem that the presence of pus in the gall-bladder occurs only after the disease has become well advanced, that gangrene of the gall-bladder usually begins at the fundus, and that a virulent peritonitis can and does occur without perforation of the gall-bladder (Robson).

The changes in the liver are comparatively slight. This seems reasonable if we accept the mechanism of the infection as described in the foregoing. The organisms would be prevented from attacking the common and the hepatic ducts on account of the smoothness of their linings and the flow of bile, while the stagnation of the bile and the presence of large folds in the mucosa would predispose the cystic duct to infection.

The general changes in the body correspond to those that accompany any acute inflammatory process.

SYMPTOMATOLOGY. The number of cases of acute primary cholecystitis is too small to outline a definite symptomatology. I shall discuss the symptoms in the order that I believe they occur, and, at the same time, shall mention the relation of the symptoms to the pathological changes.

Chill. The chill may be severe, moderate, or absent. It can be expected in the most severe cases.

Pain. The pain may accompany, precede, or follow the chill. It is of sudden onset, is very severe, is paroxysmal at the beginning, and is located in the epigastrium. It frequently radiates to the right scapular region, and in one of Richardson's cases was situated as low as the groins. The pain, in some cases, is identical with biliary colic. It is doubtless due to the gall-bladder trying to empty itself. When the musculature of the gall-bladder exhausts itself the pain ceases, only to return in a short time. When the tension in the gall-bladder becomes great and its musculature exhausted, the pain becomes continuous. When gangrene of the gall-bladder occurs the pain ceases. I shall not discuss the mechanism of acute colicky pain in the region of the gall-bladder. There are so many contradictory and radical statements on this point, made without proof, that it is impossible to draw conclusions.

Vomiting. Vomiting may be expected in most severe cases. It is an early symptom. It usually ceases after the first twenty hours, but returns if peritonitis develops. No cases of fecal vomiting have been recorded.

Fever and Elevation of Pulse Rate. These are present in all cases. They occur shortly after the patient has recovered from the chill and the shock of the primary paroxysm. The intensity of these symptoms depends on the severity of the infection. They still are present after the gangrene has abolished pain and obscured local tenderness.

Local Tenderness. This is an early and constant symptom. The entire abdomen may be sensitive, but the rigidity and tenderness

are most marked on the right side, and the point over the gall-bladder is most sensitive to pressure, except when the gall-bladder becomes gangrenous. A gangrenous gall-bladder is insensible.

Jaundice is not a symptom of cholecystitis. It may occur as a complication or as part of a pre-existing disease.

Enlargement of the gall-bladder and the liver usually cannot be determined.

Constipation. Constipation is usually present, but in most cases will yield to treatment.

The changes in the blood and urine correspond to those found in ordinary acute infections.

DIAGNOSIS. Cholecystitis is recognized by the sudden onset, the characteristic pain, the fever, and the local point of tenderness. Gangrene of the gall-bladder is strongly suspected when the pain and tenderness cease, or are markedly diminished, with the general symptoms increasing in severity. Collapse might follow perforation. Further reports will be necessary to determine this point. It is impossible to recognize gallstones when they have given no previous symptoms.

PROGNOSIS. Acute primary cholecystitis is amenable to successful surgical treatment in all cases in which operative treatment has not been unduly delayed. The mortality depends entirely upon the promptness of recognition and treatment. I am of the opinion that most of the cases would die if not treated surgically. Doubtless many patients who have died from "inflammation of the bowels" had acute primary cholecystitis at the beginning of their illness.

The subacute and the mild cases of acute primary cholecystitis recover. I believe that primary cholecystitis may occur in all degrees of severity, from the mild attacks that Musser believes to be followed by gallstones to the most severe ones that cause death in twenty-four to forty-eight hours.

TREATMENT. I know of no medical treatment for cholecystitis. If the patient refuses surgical aid, the stomach should be washed out, rectal feeding resorted to, morphine given if necessary, and cathartics avoided.

I am of the opinion that the advice of Ochsner not to operate during an acute attack of cholecystitis is dangerous. I believe that all acute cases should be operated upon as soon as the diagnosis is made. Cholecystostomy meets the indication in the early stages and is not so severe on the patient as is cholecystectomy. At present I should advise removal of the gall-bladder only in cases with extensive gangrene, and drainage of the gall-bladder in all other acute cases. The presence of stones does not modify the treatment. I believe that in acute infected cases cholecystostomy has a smaller mortality than has cholecystectomy. If the foregoing advice is followed, a secondary operation may occasionally be required.

This is not especially objectionable. Secondary operations on the gall-bladder are usually safe and are not complicated with acute infection.

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VARIETIES OF SPLENIC ANÆMIA.

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THIS report of five cases, with pathological studies in three of of them, is presented as a contribution to the knowledge of the condition generally designated as splenic anæmia. I have elsewhere expressed doubt regarding the individuality of such a condition, and the conviction that many different sorts of diseases have been classed under this name. For convenience of clinical terminology the name may, however, be retained, and perhaps some order may be established before long.

CASE I. *Male, aged fifty-nine years, history of dysentery(?), duration over two years, probably six years or more; stunted growth, pigmented skin, clubbed fingers, osteoarthritis, enlarged spleen; chloroanæmia with leukopenia, hemorrhages, diarrhœa; early enlargement followed by diminution in the size of the liver; numerous intercurrent infections; death in stupor; autopsy.*—L. C., a cigarmaker, aged fifty-nine years, native of Belgium, was admitted April 18, 1900. The history recorded at that time is as follows: His father died of phthisis, his mother of jaundice, one brother of rheumatism. He had jaundice twenty-two years before and gonorrhœa sixteen years before. He denied syphilis and malaria, but had been in Mexico and Africa with the French army and may have had malaria. He gave an uncertain account of some sort of diarrhœal

disease in Africa, perhaps dysentery. He was married at thirty-two; his wife and two children died of phthisis.

Four and a half years ago noticed a beginning alteration in the skin in the form of patches of pigmentation, alternating with light areas. Six or seven months ago had a chill and fever, and this has been repeated at intervals of a few weeks. He complains of a buzzing in his ears and of a scaly, itchy eruption on his legs. During the last two months has grown weak and dyspnœic.

FIG. 1.

Status Præsens. The patient is a somewhat undersized man. There is a bronze discoloration of legs and arms, and to a less extent of abdomen and chest, with areas of lighter color between. In places on the abdomen the skin in the bronzed areas is desquamating, leaving normal-colored skin beneath. The fingers are markedly clubbed. Patches of dermatitis are found on both ankles, and there

is slight œdema. The liver extends from the fifth rib to within an inch of the umbilical level. The spleen extends from the eighth rib to an inch below the anterior superior spine of the ilium. Two marked notches can be felt in the anterior border. Scattered rales are heard in the lungs; a systolic murmur and an accentuated second sound at the aortic interspace. Otherwise normal physical signs are obtained (Figs. 1 and 2).

FIG. 2.

April 18, 1900. Blood examination: Red blood corpuscles, 3,520,000; hæmoglobin, 51 per cent.; white blood corpuscles, 5280.

May 1*st* Blood examination: Red blood corpuscles, 3,630,000; hæmoglobin, 76 per cent.; white blood corpuscles, 2600. (The

first hæmoglobin estimate was thought erroneous at the time.) Differential count: Polymorphonuclear, 68.9 per cent.; mononuclear, 1.9 per cent.; transitional, 4.9 per cent.; lymphocytes, 22.6 per cent.; eosinophiles, 1.4 per cent.

Urine Examination. Specific gravity, 1010; acid; no albumin; no sugar; microscopically negative.

6*th.* The patient had a chill, followed by fever, delirium, and cellulitis of right leg, which finally required incision for the removal of a small amount of pus. The leukocytes numbered 6120; the blood was examined for malarial organisms at the time of this chill, and repeatedly later, but none were found. The cellulitis seemed the result of scratching one of the eczematous patches on the ankle.

June 5*th.* Slight jaundice, no change in liver, no fever.

13*th.* Right-sided orchitis; œdema of right leg.

14*th.* Chill; temperature, 104⅘°; impairment and rales at right base.

21*st.* Still impairment at right base.

24*th.* Blood: Red blood corpuscles, 3,560,000; hæmoglobin, 46 per cent.; white blood corpuscles, 2760.

July 3*d.* Considerable hemorrhage for several days, due to extraction of a tooth.

August 9*th.* Continuance of slight jaundice; also occasional elevations of temperature. No bile in urine.

September 11*th.* Marked pharyngitis.

October 17*th.* Periodic neuralgia in various parts of the body of late.

November 2*d.* Spleen seems a little smaller; liver certainly smaller.

9*th.* Definite neuritis. Arsenical treatment suspended. Improvement on discontinuing the drug. Pigmentation more marked of late.

26*th.* Another attack of lymphangitis of right leg. Inguinal glands at saphenous opening enlarged. The attack began with chill and fever. Blood examination: Red blood corpuscles, 3,710,000; hæmoglobin, 74 per cent.; white blood corpuscles, 4160.

December 17*th.* Another attack of right-sided orchitis.

January 7, 1901. Diarrhœa.

17*th.* Considerable hemorrhage after extraction of a tooth.

February 17*th.* Chill; temperature, 101°; facial erysipelas.

March 4*th.* Pain in right elbow.

19*th.* Marked pharyngitis and laryngitis. Some impairment of resonance in right chest. Mucopurulent blood-streaked sputa. No tubercle bacilli. Daily fever in afternoon. Blood: Red blood corpuscles, 4,670,000; hæmoglobin, 60 per cent.; white blood corpuscles, 1920.

May 8*th.* Another attack of right orchitis.

June 10*th.* Recently complains of pain in region of spleen.

16*th.* Much pain in right arm.

July 8th. Marked diarrhœa. Blood: Red blood corpuscles, 2,950,000; hæmoglobin, 45 per cent.; white blood corpuscles, 2720.

October 15th. Liver smaller, lower border only two fingers' breadth below costal margin. Hair growing very white. Pain in right arm continues.

22d. Blood: Red blood corpuscles, 4,060,000; hæmoglobin, 61 per cent.; white blood corpuscles, 2460. Differential count: Polymorphonuclear, 78 per cent.; mononuclear, 6.52 per cent.; transitional, 6.5 per cent.; lymphocytes, 6.5 per cent.; eosinophiles, 2.5 per cent.

November 11th. Thickening about right elbow-joint.

February 5, 1902. Blood and mucus from bowel. Fistula discovered.

March 4th. Increased diarrhœa. Patient's strength failing greatly. Blood: Red blood corpuscles, 3,160,000; hæmoglobin, 64 per cent.; white blood corpuscles, 10,600.

8th. Blood: Red blood corpuscles, 3,890,000; hæmoglobin, 68 per cent.; white blood corpuscles, 11,040. Unconscious and restless during night; died in stupor.

Autopsy. (Dr. Hendrickson, pathologist.)

Pathological Diagnosis. Pigmentation of skin and jaundice; chronic splenic tumor; atrophic cirrhosis of liver; acute lobar pneumonia; chronic interstitial orchitis; arteriosclerosis of aorta; chronic arthritis of right radiohumeral joint.

Detailed Description of the Organs. External Appearance. Body that of an extremely emaciated and poorly developed white male. Mucous membrane pale; rigor mortis absent; no œdema; considerable post-mortem lividity of dependent portions. Over sacrum is discovered a small bed-sore about 3 cm. in diameter and superficial in character. There is marked jaundice with a brownish pigmentation of the skin. The distribution of the pigment is confined entirely to the extremities. It is found over lower extremities below the knees in large, irregular patches, which are more or less confluent; the intervening areas of skin are of normal appearance. The same pigmentation is found over the hands and forearms. Fingers show marked clubbing. Superficial lymph nodes not enlarged.

Abdominal Cavity. Spleen is found enlarged and projects about 6 cm. below the costal margin. Upper border firmly adherent to the under surface of diaphragm. Liver does not extend below the costal margin except in the epigastric spaces, where it projects 3 or 4 cm. below the xiphoid process. Otherwise cavity negative. Mésenteric lymph nodes not enlarged.

Pleural Cavities. Negative.

Pericardial Cavity. Negative.

Heart. Weight, 270 grams; normal except myocardium, which is paler than it should be.

Lungs. Left: Surface dark red in color, but everywhere smooth. On section cut surface dark red and from the cut vessels considerable blood flows. Consistency considerably increased, and on pressure a marked amount of dark, bloody fluid and air escapes. The condition is uniform from apex to base. Bronchial lymph nodes deeply pigmented, but not enlarged. Right: Surface dark-red and smooth. No evidence of pleurisy. On section the lower lobe and small portion of the upper lobe are found consolidated. This part of lung is grayish-red in color, and on pressure a small amount of puriform fluid can be expressed. Middle lobe and upper portion of upper lobe are not so firm in consistency, but show marked congestion. Bronchial lymph nodes also pigmented, but not enlarged.

Spleen. Weight, 950 grams; considerably increased in size. Except where adherent to diaphragm, surface smooth. On section, capsule slightly thickened; trabeculæ prominent; pulp increased in amount and dark red in color. Malpighian bodies not visible. Consistency of the organ decreased.

Gastrointestinal Tract. Normal except at lower portion of rectum, where the mucous membrane shows marked swelling and congestion of the bloodvessels.

Pancreas. Normal.

Liver. Considerably decreased in size; surface roughly lobulated, giving rise to a typical hobnail surface. Over the anterior surface of the right lobe is found an irregular-shaped area of depression about 4 cm. in diameter, which, however, shows no thickening of Glisson's capsule. Also along the free border near the gall-bladder there is an area of depression. Section through these areas shows no scars in the depth. General appearance of cut surface is dark brownish-red, but liver tissue is divided into large areas of varying size by connective tissue. Consistency of liver distinctly increased. Gall-bladder normal.

Adrenals. Normal.

Kidneys. Of normal size; capsule strips readily; surface grayish-red in color. Weight of kidneys, 300 grams. No injection of stellate veins. Cut surface shows moderate congestion of bloodvessels, but otherwise no visible change.

Bladder and Ureter. Normal.

Prostate. Normal.

Testicle. Left, normal; right, slightly decreased in size. Slight increase in thickness of its surrounding coats. Throughout the gland proper is discovered a marked increase in grayish, firm connective tissue. Tubules do not pull out as readily from this gland as from the left.

Aorta. Shows a few scattered patches of sclerosis. None of the lymphatic glands show increase in size.

Bone-marrow. From shaft of femur is dark grayish-red in color, and of firm consistency.

Organs of Neck. Marked congestion of bloodvessels of mucous membrane over larynx and trachea. Considerable mucus in trachea. Right thyroid lobe slightly increased in size. On section, however, nothing abnormal can be discovered.

Spinal Cord. Examination without sectioning shows nothing abnormal.

Right radiohumeral joint removed *in toto.* Examination of the interior of this joint shows a superficial erosion of cartilage and bone over a small portion of the prominence separating the radial articular surface from adjacent bone. Also a more marked erosion of the articular surface in the greater sigmoid cavity of the ulna.

FIG. 3

Coarse hæmatogenous pigment masses surrounded by fibrous tissue.

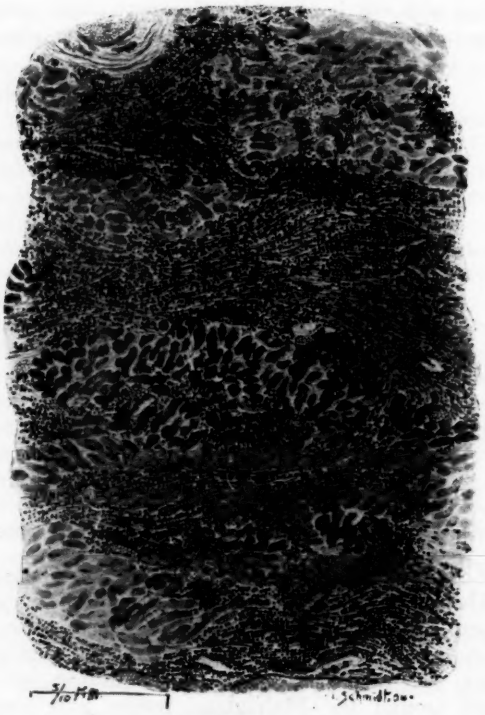
Microscopic Features. (Examination by Dr. Stengel.) *The Spleen.* There was throughout this organ a diffuse connective-tissue hyperplasia, involving the walls of bloodvessels, the trabeculæ of the organ, the splenic pulp, and the Malpighian bodies. Throughout the pulp this process was diffuse and cellular, but in and around the Malpighian bodies and elsewhere in circumscribed foci it was of a denser nature with a marked tendency to hyaline change. In a number of places a peculiar form of pigmentary deposit was observed within areas of dense fibroid tissue. In such areas brownish pigment masses of a hyaline or almost crystalline appearance were found within a focus of dense connective tissue. Stained with ferrocyanide of potash and muriatic acid these formations gave a distinct iron reaction, which, with the general appearances, showed the pigmentation to be of hemorrhagic origin. The coarse character of these lesions and the marked connective-tissue change around them gave a peculiar appearance that

I have not seen in any other spleen, excepting from one other case of similar clinical character (Fig. 3).

The connective-tissue growth in the walls of bloodvessels and surrounding them was very pronounced, and many of the smaller vessels were entirely obliterated.

In the splenic pulp many pigment cells containing rather coarse, brownish pigment were found, quite apart from the areas of marked

FIG. 4.



Section of liver. × 41.6.

pigmentation before referred to. Everywhere throughout the spleen there were foci of recent hemorrhage, sometimes side by side with older hemorrhagic pigmentation, and sometimes independent of such. Altogether there were decided evidences of congestion and hemorrhagic processes of old and recent origin with secondary fibroid and pigmentary changes. Independent of these the substance of the organ presented a diffuse fibroid change of seemingly irritative character. Under higher powers swollen and

proliferated endothelial cells were moderately conspicuous in the sinuses, but nowhere were these aggregated to form distinct masses.

The Malpighian bodies were extensively altered. Many had become converted into fibrous and hyaline masses; others were partially affected by fibrous change beginning around the central vessel; only a few were found without some alteration of structure.

The Liver. There was a very pronounced grade of connective-tissue infiltration of interlobular and intralobular character. In many places the hepatic lobules were surrounded by a dense cellular connective-tissue growth which was most markedly interlobular, but also invaded the atrophied lobules. The liver cells themselves were moderately pigmented, but were quite generally distorted and atrophied. The portal veins were widely distended, but the central vessels of the lobules were narrowly contracted and nearly free of blood (Fig. 4).

The Lymphatic Glands. All of the lymphatic glands showed diffuse, fibrous change of moderate character, and intense congestion with widely dilated blood spaces. The glands from various portions were not examined with reference to the distinctive changes in the different groups.

The Bone-marrow. The marrow from the shaft of the femur showed distinct lymphoid characters. The majority of the cells were large and small neutrophile myelocytes, with pale, eccentric nuclei. Eosinophile myelocytes were present in small numbers, and rather more eosinophile cells of the ordinary type. Some pale hæmoglobin-containing cells of doubtful nature were observed, but no distinct nucleated red corpuscles. Polymorphonuclear cells and fat cells were not numerous.

Other Organs. The sections of the lung showed decided fibroid and cellular pneumonia. There was undoubtedly an old fibroid process with more recent bronchopneumonia. The pancreas and kidney showed moderate sclerotic change. The right testicle was markedly altered, being practically converted into fibrous tissue. Several areas of hemorrhagic pigmentation, similar to the coarse pigmentation in the spleen, were found in the testicle.

CASE II. *Female, aged two years and eight months. Enterocolitis at one year; little evidence of rickets; pallor, enlarged spleen, palpable superficial lymph glands; severe anæmia, at first without, later with leukocytosis; diarrhœa, terminal tuberculosis, and endocarditis; duration one and a half years; autopsy.* Elsie S., aged two years and three months; admitted October 13, 1899.

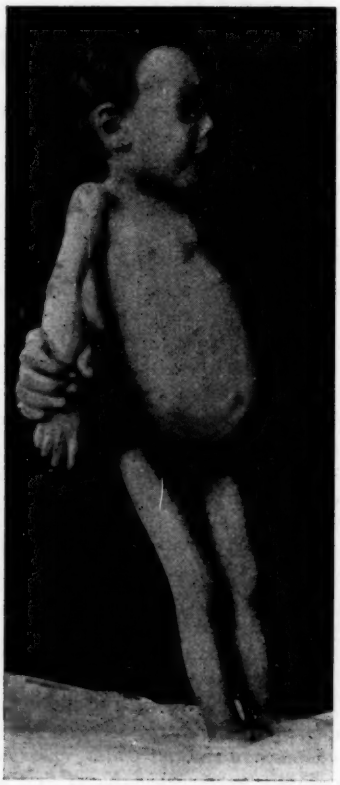
Family History. Both parents and two sisters are living and in good health.

Previous History. One year before had a severe attack of enterocolitis. The child was healthy with this exception until the present illness. Began to walk when twelve months old (Fig. 5).

Present illness began when she was twenty months old. The parents first noticed increasing size of the abdomen and pallor.

Status Præsens. Ashy pallor; slight osseous signs of rickets; abdomen large and globose, quite tense. Enlarged spleen, with notches easily palpable. Liver enlarged to percussion, but edge cannot be felt. No fluid in abdomen.

FIG. 5.



All superficial lymph glands enlarged and easily palpable. Heart is found under sternum, extending an equal distance to right and left. A short systolic, puffing murmur is heard at the left side of cardiac dulness. Breath sounds harsh, but resonance good.

Blood Examination. Red blood corpuscles, 2,920,000; hæmoglobin, 30 per cent.; white blood corpuscles, 7600. Differential count: Polymorphonuclear, 50 per cent.; mononuclear, 17 per cent.;

lymphocytes, 31 per cent.; eosinophiles, 1.6 per cent.; myelocytes, 0.4 per cent.

There was constant diarrhœa, the movements numbering from three to five daily.

October 25, 1899. The condition continues without much change. The skin has become somewhat yellowish. At this date the spleen extends a finger's breadth below the anterior superior spine of the ilium, and the anterior edge is within one-half inch of the middle line. The liver is still enlarged to percussion, but the edge cannot be felt.

The heart is now in a more normal position, extending from the third rib above to the sixth below and from left nipple line to right border of sternum. The same soft, systolic murmur is heard, but it is not transmitted beyond the cardiac borders. The point of maximum intensity is at the pulmonary region. The second pulmonary sound is not accented.

The left kidney can be plainly felt in the back, being seemingly pushed backward; the right is less plainly felt.

26*th*. Several small masses can be felt in the abdomen. They seem to disappear after a movement of scybalous masses.

November 4*th*. The submaxillary, submental, and cervical glands are distinctly enlarged.

15*th*. Moderate fever and continued diarrhœa.

December 8*th*. General urticaria followed by swelling of eyelids, face, legs, and arms.

10*th*. The swelling has disappeared.

11*th*. Blood: Red blood corpuscles, 3,920,000; hæmoglobin, 40 per cent.; white blood corpuscles, 14,720. Differential count: Polymorphonuclear, 49.2 per cent.; mononuclear, 2.4 per cent.; transitional, 12.8 per cent.; lymphocytes, 34 per cent.; eosinophiles, 1.6 per cent. Polychromatophilia; a considerable number of normoblasts.

16*th*. Physical signs the same, but liver seemingly larger. Edge can be felt. Kidneys still easily palpable in flanks.

January 27, 1900. Has been better of late. Now a greenish-yellow pallor is noted.

February 15*th*. Sudden fever, with scarlet flush of face and neck; later, papules which were bright red at first, but soon faded.

The later notes have been unfortunately mislaid, but the final phase was one apparently of disseminated pneumonia with continued fever of about 103° to 104°; respirations varying from 50 to 68, and corresponding pulse rate.

(The autopsy was made by Dr. Flexner.)

Pathological Diagnosis. General lymphatic enlargement, with congestion and hemorrhage of the glands; splenic tumor, with infarctions; anæmia of the organs; subacute, vegetative, mitral endocarditis; necrosis of the mucous membrane of the œsophagus; tuber-

culosis of the lymphatic glands and tuberculosis of the lungs; infarction of kidneys.

Bacteriological Diagnosis. Staphylococcus aureus from heart's blood; aortic valves; œsophagus; lungs.

External Appearance. Body of an emaciated, very pale child. Length 82½ cm. The abdomen is much distended. No evidence of fluid. The spleen can easily be felt in the left hypochondriac region. Its lowest border is 21 cm. in the midaxillary line below the ribs. The circumference of the abdomen below the ribs is 47½ cm., and just above the umbilicus 43½ cm. There is a very small amount of subcutaneous fat; indeed, it is in most places absent. There is very slight œdema of the lower borders of the ribs. Over the extremities, particularly the lower extremities, are minute, petechial hemorrhages. Over the abdomen are several pigmented depressions, which are probably the remains of small furuncles. No rigor mortis.

Abdominal Cavity and Peritoneum. Upon opening the abdominal cavity the omentum is seen to be adherent to the greatly enlarged spleen. Upon being stripped back, which can be done in part only on account of the old adhesions, it is found that there is a fresh exudate of yellow fibrin over the upper part of the enlarged organ. In the lower border there is an infarction about 2 cm. in width. The surface of the spleen is mottled, the predominating color being brown, and lighter areas corresponding to other infarctions can be seen on its superficial surface. There are no adhesions, excepting to the diaphragm on the upper edge, with the exception of those to the omentum as already described. The spleen weighs 465 grams. Its measurements are 17 x 9 x 5 cm. Its consistence is firm and even very hard. On section the infarctions described are found to be on the anterior surface; they are three or four in number, varying from 1½ to 3 cm. in their widest portion. They are either anæmic or mixed in character. The mixed ones show a cortical anæmic layer and a deep brownish-red, rather soft, central portion. They extend about 2 cm. into the substance of the organ. In other portions the spleen is of a reddish-brown color, and there is no distinction of Malpighian corpuscles. Here and there on the cut surface are whitish points about the size of pinheads, which are taken to be small, dense thrombi.

Liver. With the exception of an adhesion on the anterior surface of the right lobe to the abdominal wall, measuring 1½ cm. in diameter, and very old in nature, it is free from adhesions. Its weight is 690 grams. Its cut section is cloudy. Its consistence is not increased.

Kidneys are alike. The capsule strips off easily, showing in each organ a recent infarction about 4 mm. in diameter in the cortex, about which the tissues are greatly congested or hemorrhagic. On section the cortex is pale, pyramids somewhat more injected. Be-

neath the mucous membrane of the pelvis there is a reddish accumulation which looks like tissue, or may be hemorrhage. Adrenal glands appear normal.

Heart and Pericardium. Pericardial cavity contains about 30 c.c. of clear, yellow fluid. The layers of the serosa are smooth. The right ventricle is dilated. The left ventricle is contracted. The heart's flesh is pale. The tricuspid, pulmonary, and aortic valve segments are delicate. The mitral segments are covered with vegetations, quite adherent, which average in size about a bird-shot, some being larger, and as large as a hemp-seed. The two edges of the valve are rather difficult to separate, but this union is due probably to a small clot formed post-mortem over the edge. In the diaphragm there are scattered whitish points, probably tubercles.

Lungs. The right lung is bound over the upper portion of the upper lobe to the chest wall. The left lung is bound over the apex to the chest wall. There is no fluid in the pleural cavities. The lungs are insufflated. On section the upper lobes contain disseminated, gray tubercles; fewer in the lower lobes. There are no definite foci of consolidation, although in the pleura over each upper lobe are small hemorrhages. Beneath the parietal pleura on the left side, and following the intercostal muscles, are found white or caseous areas of tuberculosis. In the bifurcation of the trachea there is a large gland, the size of a pigeon's egg, which upon section is completely caseous.

Stomach and Intestines. The stomach shows nothing of special interest. Post-mortem digestion. The duodenum contains a number of elevated, swollen, solitary follicles, after which there is no swelling of any of the follicles throughout the length of this intestine. In the lower part Peyer's patches are visible, but nothing more. The mucous membrane of the large intestine is attenuated. In general, the intestine is thin and apparently atrophic.

Tonsils. Enlarged, and there are irregular depressions. No definite foci can be made out. In the upper part of the œsophagus, on a line with the upper part of the thyroid cartilage, there are two foci of necrosis, each about 4 mm. in width, over which there is a dirty-white exudate.

Lymphatic Glands. The inguinal, axillary, and cervical glands can easily be made out by palpation. The glands of the neck cause the tissue on each side to project. All the lymphatic glands of the body, upon examination, are found to be enlarged and to be very red in color. The greatest enlargement is found in the cervical and in the inguinal regions, in both of which places the superficial as well as the deep sets are swollen. The swelling affects the individual glands and not the invading tissue. In the cervical region the largest gland is about the size of a pigeon's egg. Most of them vary from a pea to a cherry in size. In the inguinal region the largest

is about the size of an almond. The retroperitoneal glands form chains and large, red bodies. The mesenteric glands and the glands in the mesocolon and the omentum are all enlarged and congested or hemorrhagic. The glands about the pancreas are especially enlarged. Some of them contain whitish nodules, evidently tubercles. The largest of the mesenteric glands contain also a white area, suggesting tuberculous caseation.

Bone-marrow. The bone-marrow of the femur is abundant and red in color. Its consistence is soft.

Microscopic Features. (Dr. Stengel.) The sections of the spleen varied considerably in their appearances according to the part of the organ from which they were taken. Those from the areas of infarction showed only the usual features of dense hemorrhagic infiltration and pigmentation. In other parts the splenic tissue was markedly sclerotic, and sections stained with Biondi's or Van Gieson's stain presented a uniform, rather dense, fibrous reticulum. Hyperplasia of the endothelial cells of the sinus was evident in some places, and large (swollen) endothelial cells of rounded or irregular shape, with one or a few pale nuclei, were found scattered through the spaces. In a few areas groups of such cells were seen lying together. The proper splenic pulp and the Malpighian bodies had undergone an evident atrophy, and many of the latter were converted into hyaline material. A few doubtful miliary tubercles were seen in the sections. These consisted of a small giant cell surrounded by a group of round (lymphoid) cells. There was no evidence of necrosis.

Lymphatic Glands. The sections of mesenteric lymph glands showed marked thickening of the capsules and of the trabeculæ, the latter separating the lymph follicles in an unusually marked manner. The medullary part of the glands was occupied by numerous widely distended blood spaces or vessels. The lymphoid tissues proper showed no special alteration, but decided proliferation of endothelial cells of the sinuses was seen in places, and numerous macrophages containing one, two, three, or more erythrocytes or fragments of such were found. The red corpuscles within the lymph sinuses were much broken, and everywhere fragments of irregular or more commonly rounded shape, and with distinct basic staining reaction, were found among the degenerated erythrocytes. These fragments were of precisely the appearance of blood plaques, and from their situation and general character could be plainly recognized as derivatives of degenerated red corpuscles.

The Liver. Marked lymphoidal and leukocytic infiltration between the columns of liver cells was the most striking feature of the sections as seen under a low form. In areas there was distinct interlobular fibrosis surrounding the portal vessels. The latter were considerably dilated and, besides containing fragmented and degenerated red corpuscles as well as normal erythrocytes and leukocytes, in many places also contained numerous plaque-like forma-

tions such as have been described for the lymphatic glands. Here and there in the dilated portal vessels could be found large endothelial cells of the same appearance as those of the spleen and lymphatic glands. These were distinguishable from the lining endothelium of the bloodvessels of the liver, and if derived from this source had manifestly undergone secondary change. No such cells could be found outside of the bloodvessels. They were too numerous and widespread to be attributed to accident or artefact.

The Lung. The lung tissue in general showed no marked alterations in the sections examined, but a few peribronchial miliary tubercles were seen in portions especially selected to determine the nature of the gray bodies observed at the autopsy.

The Bone-marrow. Unfortunately the material was lost and microscopic examination could not be made.

CASE III. *Female, aged twenty-one years, married, two stillborn children; probable onset years before; enlarged spleen, palpable superficial lymph glands, pain in left side, slight anæmia, splenectomy; postoperative pneumonia; recovery; histological examination of spleen.*—Annie D., aged twenty-one years, negro; lived in North Carolina until a year ago, when she came to Philadelphia. Had two stillborn children about a year ago. Her father died of malaria, her mother of an accident. No brothers or sisters. The patient has always been in good health; menses always regular and normal. She says that for many years there has been more or less pain in the left side of the abdomen. She cannot tell exactly how long this has been. Until three months ago the pain was not, however, severe enough to keep her from attending to her usual duties, but during the last three months it has been so severe at times as to compel her to take to bed. During the same length of time she has noticed a lump in the left upper abdominal quadrant. The pain is dull and aching in character, but becomes more severe when the patient moves about. There has been some loss of weight during the last three months.

Status Præsens. The patient is fairly well nourished, looking somewhat older than her years. There is nothing striking on external examination excepting a projection in the left upper half of the abdomen. The superficial lymphatic glands, cervical, axillary, inguinal, and epitrochlear, are all palpable. Heart and lungs normal. Occasionally a short systolic murmur was heard over the root of the pulmonary artery.

Abdomen. There is marked prominence of the left side above. This moves downward on full inspiration. On palpation a greatly enlarged spleen could be palpated and was distinguishable by the notches in front. At first palpation was somewhat difficult on account of tenderness, but later the patient seemed to be suffering less pain. The mass extended downward and forward to within an inch and a half of the linea alba at the level of the umbilicus, and

to about the level of the crest of the ilium below. The posterior border is felt as a thick, rounded surface, while the anterior border has a fairly sharp edge with a distinct notch. No nodules or irregularities can be felt along the edge above the ribs. The mass extended to the sixth rib. The liver extended from the sixth rib to the edge of the ribs. It was not palpable. The stomach was crowded upward and forward, as could be determined by inflation, and the colon was pushed downward.

Blood. Red blood corpuscles, 4,440,000; white blood corpuscles, 5600; hæmoglobin, 70 per cent. The stained specimens show a slight reduction in the color of the red blood corpuscles, but no other peculiarities were discovered. Degenerative changes, such as polychromatophilia and basic degeneration, were wanting. The differential count of the leukocytes at this time showed no marked peculiarities, though the count has been lost.

Urine. Specific gravity, 1020; amber color, acid, no albumin, no sugar. Microscopically granular precipitate, epithelial cells, and a few leukocytes were found.

The patient was kept under observation for some weeks, during which the condition remained practically unchanged. There was apparently no increase in the size of the spleen and no change in the patient's general condition excepting perhaps a decrease in the amount of pain. Her weight remained exactly the same. It was decided that the case was one of splenomegaly and that, on account of the pain and incapacity of the patient, operation was desirable. For this purpose she was returned to the service of Dr. John G. Clark, who originally had referred her to me, and who has kindly permitted me to report the case.

On the day preceding the operation the differential count showed the following figures: Lymphocytes, 29 per cent.; mononuclear, 7.4 per cent.; transitional, 5.4 per cent.; polymorphonuclear, 50 per cent.; eosinophiles, 7.8 per cent.; basophiles, 0.4 per cent. The subsequent leukocytic counts following the operation will be seen in the table on the opposite page.

The operation of splenectomy was performed by Dr. Clark on January 28, 1904. The following notes were recorded at the time: The spleen was rather dense in consistency and the surface showed numerous nodules about 1 to 2 cm. in diameter, with depressed centres. The relations of the organ were found as described in the physical examination. There were a few adhesions posteriorly and some in front, between the lower border of the tumor and the great omentum. These were tied off; the others gave no trouble. The splenic vessels were ligated with some difficulty on account of the very narrow space between the tail of the pancreas, the cardiac end of the stomach, and the spleen. The liver was slightly enlarged and showed a number of scar-like depressions on the surface of both lobes.

Date.	Red blood corpuscles.	Hæmoglobin.	Leukocytes. Total count.	Lymphocytes.		Mononuclear.		Transitional.		Polymorphonuclear.		Eosinophile.		Basophile.	
				%	No.	%	No.	%	No.	%	No.	%	No.	%	No.
Jan. 1, 1904	.....	.....	5,600												
" 27, "	.....	.....	.....	29.0	.....	7.4	.....	5.4	.....	50.0	.....	7.8	.....	0.4	
" 29, "	.....	.....	.....	5.0	.....	4.2	.....	4.0	.....	86.5	.....	0.2			
" 30, "	3,750,000	55 %	35,000	3.0	1050	4.4	1540	7.6	2660	84.8	29,680	.....	.....	0.2	70
" 31, "	.....	62 %	16.640	5.0	832	2.5	416	10.2	1697	74.4	12,380	3.6	599	0.5	85
Feb. 1, "	3,350,000	.....	12,720	3.4	432	2.4	305	8.2	1043	74.8	9,514	10.5	1335	0.3	38
" 2, "	.....	.....	21,120												
" 3, "	.....	.....	21,760												
" 4, "	4,190,000	55 %	17,680												
" 6, "	.....	.....	21,120												
" 10, "	.....	.....	20,000	6.6	1320	2.2	440	7.8	1560	81.0	16,200	2.4	480		
" 14, "	3,840,000	.....	21,520												
" 16, "	.....	.....	23,860												
" 18, "	.....	.....	9,600	9.0	864	2.6	250	9.6	921	70.7	6,787	7.9	758	0.2	19
" 24, "	.....	.....	9,000												
Mar.14, "	4,030,000	55 %	9,120												
May 5, "	4,900,000	55 %	7,280	18.6	1354	2.8	204	5.7	415	61.9	4,506	11.0	800		

Almost immediately after the operation the patient complained of pain at the base of the left chest and in the left upper part of the abdomen. The temperature rose to 102° and subsequently kept constantly above normal. At first physical examination was difficult because of the impossibility of turning the patient, but indefinite rales or friction sounds were heard. Later decided signs of a lobar pneumonia were detected. Before this time, however, the upper angle of the incision was opened in the effort to discover the cause of the pyrexia, and a small area of induration, but no suppuration, was discovered. On February 17th, twenty days after the operation, and after the first elevation of temperature, the fever subsided by crisis, going from 103° to the normal in the course of a few hours, and remaining normal continuously thereafter. At the same time the leukocytes declined from over 20,000 to 9600, and subsequently remained normal or subnormal.

After the full recovery from the operation the patient showed no change in appearance or physical signs, excepting those referred to the left upper part of the abdomen. She complained of some dragging and other unpleasant sensations in this region, but as compared with her condition before the operation was certainly much relieved.

Three months after the operation I found her in comparatively good health, able to attend to her work as a chambermaid, and with practically no local symptoms. The lymphatic glands seemed to have increased somewhat in size, but the difference was not very marked.

Among the interesting features in the blood count of the case was the behavior of the lymphocytes and eosinophile cells. It has been found by experiment, as well as after operative removal of the

spleen in man, that there is an increase in the number of lymphocytes and later an increase in the eosinophile cells. The proportion of the latter was at the time of the operation decidedly increased (7.8 per cent.). The lymphocytes were not specially increased (29 per cent.), but the polymorphonuclear cells were actually and relatively reduced in number. This reduction was, however, in favor of the mononuclear and transitional cells, which at this time and subsequently showed conspicuous increase.

FIG. 6.

Spleen. × ⅓.

Unfortunately the intervention of an acute pneumonia altered the figures that probably would have been obtained. The result of the postoperative pneumonia are the same as is usually seen in normal persons—a marked polymorphonuclear leukocytosis and a reduction of eosinophiles. After the subsidence of the pneumonia there was a return to practically the same conditions as were observed before operation, viz., a marked eosinophilia and reduc-

tion of polymorphonuclear in favor of the mononuclear and transitional cells.

Macroscopic Appearance of the Spleen. The organ measured 22 x 13 x 7 cm., and after hardening weighed 1230 grams. The striking features when seen in the fresh state were numerous nodular projections on the surface, which presented the appearance of secondary tumors. These varied in size from small masses a half to 1 cm. in diameter up to large nodes fully 2½ or 3 cm. in diameter. The larger nodes were distinctly umbilicated, the depression being quite marked and of a whitish, cicatricial appearance at the bottom. Elsewhere the surface of the organ was red in color, somewhat modified by a more or less thickened capsule. The project-

FIG. 7.

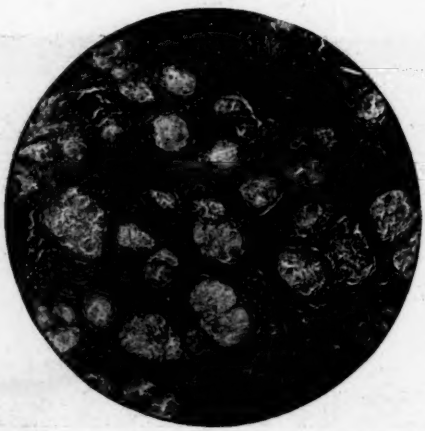
Macroscopic appearance of section (photograph). The lighter areas represent new-growth; the darker areas are uninvolved splenic tissue.

ing nodules were numerous anteriorly and on the convex surface of the organ; they were fewer posteriorly and in the hilum. The consistence of the organ as a whole was distinctly increased and was uniform in all portions. On section through its substance practically the entire organ was seen to have been invaded by a growth of a dense consistence and pinkish-red color. This extended inward and toward the hilum, leaving only small parts of the substance of the spleen, which could readily be distinguished by their darker color, uninvolved. Posteriorly and at the hilum the spleen substance was comparatively free. As seen in the cross-section the nodular masses on the surface were manifestly not separate new-growths like secondary tumors, but projecting parts of a large invading mass which occupied the greater part of the organ. The

appearance was not unlike such a one as might result from separate pyramidal growths, with the apices projecting from the surface of the organ and extending deeply into the substance, the bases being completely fused or at most leaving small areas of normal splenic tissue here and there between them (Figs. 6 and 7).

Careful examination of the umbilications of the nodules presented no evidence that these had resulted from secondary softening of the centres of the growths, but suggested rather that the elevation surrounding the depressions was the result of an infiltrating process that had not affected the centre so that the fibrous tissue of trabeculæ had prevented the elevation at the point of umbilication. Cross-sections showed some excess of fibrous tissue in this central

FIG. 8.



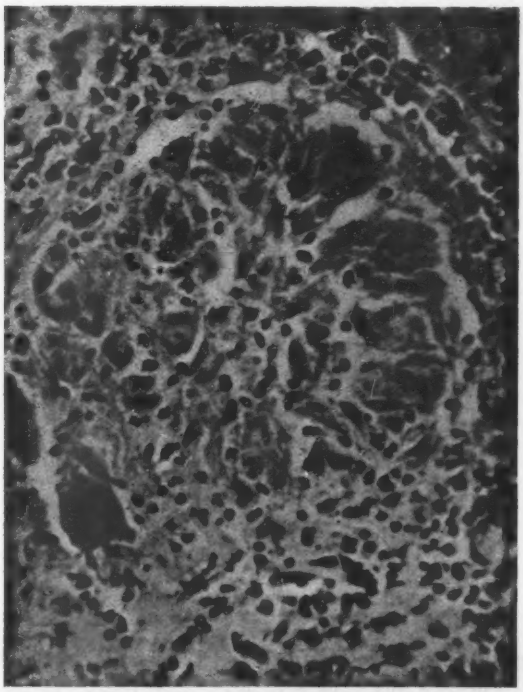
Microphotograph of section of spleen under low magnification, showing the alveoli containing endothelial cells surrounded by fibrous walls. The photograph exaggerates the density of the fibrous tissue.

zone, and the cicatricial appearance on the surface was due to thickening of the capsule of the spleen at the point noted, quite as much as to actual fibrous tissue formation in the centre of the nodes. Nowhere in the substance of the new-growth could any evidence of necrosis be discovered by the naked eye, no areas of softening, of caseation, or the like. The tissue was uniform in character, somewhat glistening on section and, as before stated, pinkish-red in color.

Microscopic Appearances. Portions of various parts of the organ were placed in Zenker's fluid, Müller-formol, and absolute alcohol, and sections were stained with eosin-hæmatoxylin, Van Gieson's stain, Biondi's mixture, carbol-fuchsin, methylene blue, and by Gram's method.

The sections showed a marked new growth of connective tissue forming alveoli of various sizes (from 0.13 to 0.26 mm. in diameter), and containing large, clear cells, with pale nuclei, and often cells containing several nuclei of the same character. In certain areas giant cells with centrally or eccentrically clustered nuclei were abundant, and everywhere such cells were present, at least in small numbers. The lymphoid elements of the splenic pulp were an

FIG. 9.



Microphotograph (high power), showing one of the alveoli containing endothelial cells, giant cells, and leukocytes.

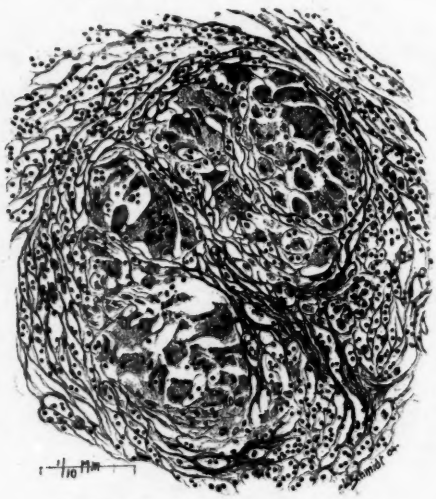
inconspicuous feature in all the sections, being crowded into mere zones surrounding the alveolar formations where these were well developed, and being displaced by more diffuse connective-tissue growth in other places.

The alveolar spaces referred to were generally rounded in outline and surrounded by a wall of well-developed connective tissue of considerable thickness. For the most part this wall was compact

and separated from the surrounding alveolar or splenic tissue by a sharp line of separation, but elsewhere the transition was more gradual and formed by connective tissue of a less dense character intermixed with lymphoid elements (Figs. 8, 9, 10 and 11).

Within the alveoli were found the large endothelial cells before described. In sections through favorable parts of the growth the cells were separate and clearly marked, but more commonly there was an apparent or real confluence making large protoplasmic structures containing large vesicular nuclei. These almost invariably showed nucleoli. Many of the cells contained two or three nuclei. In the peripheral parts of the alveoli, and to a less extent in the

FIG. 10.



Section of spleen. Biondi's stain. × 142.5.

centre, giant cells with clusters of nuclei were common. The protoplasm of the cells was clear and rather glistening. It did not present any appearance of degeneration, though in some places it was possibly somewhat hyaline. In some of the alveoli, especially those with less well-developed walls, the endothelial cells were separated by or intermixed with erythrocytes or lymphoid cells, but there were no macrophages. Pigmentation was practically absent in this part of the tissue, but was present to a small extent in the reticular connective tissue.

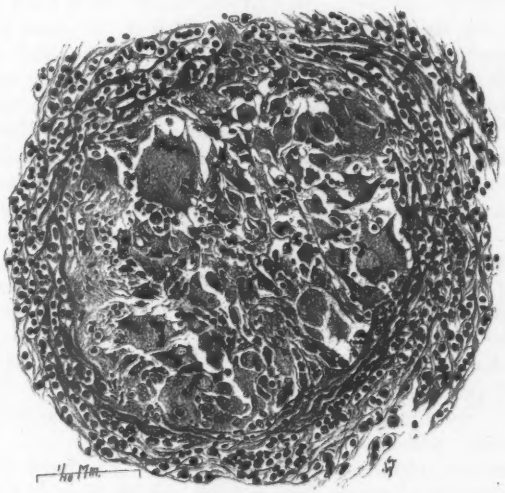
The size of the endothelial cells containing one or two nuclei varied from about 10μ to 20μ in diameter, the average being about 15μ; the size of the giant cells was from 30μ to 72μ, the average of

a number being 51μ. The large masses of fused character were even larger, in some places nearly filling the alveolus.

In the portions of the spleen comparatively free from the uniformly infiltrating growth there was still evidence of an active endothelial proliferation, but as the organ was practically entirely involved it cannot be determined if this process were simply an extension from the growth proper or an evidence of prior involvement in lesser intensity of a condition that caused complete transformation in the parts that have been designated the growth proper.

The Malpighian bodies were retained in some places, though atrophied. Elsewhere they were converted into connective tissue

FIG. 11.



Section of spleen. Mallory's stain. × 157.

or had become hyaline. This, however, was an inconspicuous feature. In general the endothelial and fibrotic process had simply caused a disappearance of normal splenic features.

In sections from one part of the spleen the Malpighian bodies were conspicuously involved, being occupied by a rounded, acinus-like area of endothelial proliferations, lying adjacent to the blood-vessel of the Malpighian body and sometimes pushing the vessel to one side, but never involving it. In some parts the area of endothelial character was minute; elsewhere almost the whole Malpighian body was transformed. There was no evidence of fibrous-tissue formation in these areas.

Sections stained with carbol-fuchsin and Gabbett's solution

showed *no tubercle bacilli*, and those stained by Gram's method likewise showed no bacteria of any kind.

CASE IV. *Man, aged fifty-one-years; history of malaria, stunted growth, pigmented skin, clubbed fingers and toes, enlarged abdomen (tympany), enlarged spleen, slightly enlarged liver, moderate anæmia, leukopenia, diarrhœa, hemorrhages from bowel, rectal ulceration, tendency to chills, no malarial organisms; duration at least three years, probably longer.*—C. T., aged fifty-one years, laborer on railroad, a native of Italy, had come to America six years before. Admitted to Pennsylvania Hospital September 30, 1901.

Family history negative. He himself had had some operation for a mass in the right inguinal region forty years before. During ten years after he was twenty years old he had mild attacks of chills and fever every summer. At thirty-three had some kind of bloody diarrhœa for six weeks. Six weeks ago first had pain in the left side of abdomen and distention of the abdomen and diarrhœa. The stools were free of blood, but the patient had much rectal pain.

On physical examination the patient was found a small-sized man with darker skin than the average Italian and distinct pigmentation in places. The fingers and toes showed clubbed extremities. The abdomen was enlarged and tense, but did not contain free fluid. The spleen could be felt extending far down into the abdomen. The liver dulness was small in vertical extent. Later the abdominal distention subsided and the spleen was then found extending to within an inch of the anterior superior spine of the ilium. Rectal examination showed an ulceration in the anterior wall of the rectum (Fig. 12).

Blood Examination. Red blood corpuscles, 4,190,000; hæmoglobin, 60 per cent.; white blood corpuscles, 4000.

Urine. Specific gravity, 1012; acid; some albumin present; granular and epithelial casts.

The sputum contains no tubercle bacilli. The feces contain no amœbæ; the blood serum does not agglutinate Shiga's bacilli.

March 5, 1902. Chill, temperature of 104$\frac{1}{5}$°; tongue greatly swollen. Later, ulceration under tongue. Leukocytes 11,400. No malarial organisms.

April 1st. Admitted to University Hospital. Same conditions noted; also enlargement of practically all superficial lymph glands. Liver extends from sixth rib to two inches below ribs. Edge easily felt. Spleen as before. A small ventral hernia.

4th. Blood: Red blood corpuscles, 3,980,000; hæmoglobin, 70 per cent.; white blood corpuscles, 4400.

10th. Hemorrhage from bowel; diarrhœa.

June 6th. Hemorrhage from bowel, about eight ounces; diarrhœa.

7th. Considerable hemorrhage from bowel; chills, but no fever.

August *12th.* Slight hemorrhage, followed by temperature of 102$\frac{2}{5}$°, soon normal.

From this date no notable changes. The patient frequently complained of abdominal pain near the umbilicus (ventral hernia), in the left side (spleen), and in the rectum (ulcers). Repeated examinations showed persistence of the ulcers. At intervals he had accessions of cough, with rough breathing and rales. The blood was examined repeatedly and was found in about

FIG. 12.

the same condition as above recorded. Constant diarrhœa and frequent hemorrhages were the marked symptoms. The patient was transferred to the Philadelphia Hospital.

This case in its general appearance and in the tendency to repeated infections resembled Case I. very closely. Studies of the blood serum were made to determine if any explanation of the tendency to infection might be found. Dr. J. S. Evans, who conducted

these examinations, reported an increase rather than reduction in both immune bodies and complements. The mechanism of the infections must therefore be sought in some other direction.

CASE V. *Woman, aged forty-four years; doubtful history of malaria, enlargement of spleen, succeeded by evidences of hepatic cirrhosis (ascites); no anæmia, hydrothorax; duration two and a half years or more.*—Ellen D., aged forty-four years, white, married. Was admitted to the University Hospital November 24, 1903. The following history was recorded:

The patient is a widow and has five children, all of whom are well. She has worked hard all her life.

The family history is negative excepting that one brother died of consumption. No history of new-growths.

Menses began at thirteen years, always normal. Labors normal. The last one eighteen years ago. There was some uterine trouble six years ago and she was confined to bed for six weeks. She had malaria two years ago, but her description of the symptoms is not very convincing.

About two years ago the patient first noticed some enlargement on the left side of the abdomen. This continued without any local symptoms excepting a slight feeling of fulness and occasionally moderate pain. There has been no loss of weight, but she has become weaker. Her color has at times been a trifle sallow. A little swelling of the feet has recently developed. This comes on during the day and disappears at night. Physical examination showed slight pallor and a subicteric hue of the skin, but no distinct jaundice. Very slight œdema of the legs and feet. No cyanosis. Lungs normal, excepting that the breath sounds appear a little harsh. Heart normal in size; the first sound at the apex is a little lacking in muscular quality, but there are no murmurs. The abdomen is enlarged, especially on the left. The spleen can be easily felt, and on the anterior surface are several distinct notches. The lower border extends below the umbilicus.

The liver extends from the sixth rib to below the costal margin. As the abdomen is generally distended it cannot be plainly felt beneath the ribs, but in the epigastric spaces the edge is distinctly palpable and the substance of the organ is evidently hard and rough. There is decided movable dulness and fluctuation in the peritoneum.

Examination of Blood. Red blood corpuscles, 5,500,000; white blood corpuscles, 8800; hæmoglobin, 81 per cent.

Urine. Clear, brownish-red; specific gravity, 1025; no albumin, no sugar; urates, a few leukocytes, and epithelial cells.

November 28, 1903. Paracentesis of the peritoneum was performed and eleven pints and three ounces of clear fluid, yellowish-green in color, were removed. The liver could then be plainly felt and was decidedly hard.

30*th*. Some pain at the right base and a pleural friction sound. Blood: Red blood corpuscles, 5,300,000; white blood corpuscles, 7200; hæmoglobin, 80 per cent.

December 1*st*. Considerable reaccumulation of abdominal effusion. Right-sided hydrothorax.

5*th*. Increase in effusion in right chest; white blood corpuscles, 7400. Urine: Specifie gravity, 1030; small amount of albumin, leukocytes, a few red blood corpuscles, urates, a few hyaline casts. There is a trace of bile.

16*th*. Abdomen tapped; twelve pints fourteen ounces removed.

January 3, 1904. Abdomen tapped; sixteen pints fifteen ounces removed. The fluid does not contain bile.

14*th*. Abdomen tapped; eighteen pints eight ounces removed. Right pleural cavity tapped; fourteen ounces of blood-tinged fluid removed. Soon after this the patient passed out of observation.

The most interesting feature in this case was the fact that she had been under my observation a year previous to her admission to the hospital, at which time I found great enlargement of the spleen without a single symptom of consequence. It was not until long after that time that she first showed evidences of beginning cirrhosis and portal obstruction. There was not in the history of this case any hemorrhage nor the usual gastrointestinal symptoms met with in splenic anæmia.

Classification of Splenic Anæmia. Analysis of the clinical descriptions and pathological features of many of the cases classified as splenic anæmia discloses the fact that these cases properly belonged to one or the other of the following conditions:

1. *Progressive Pernicious Anæmia or Intense Secondary Anæmia.* The oft-cited case of Strümpell belongs to this group. In that case, probably one of pernicious anæmia, simple congestion with great dilatation of the splenic sinuses constituted the pathological condition of the spleen. Other cases in which pronounced anæmia and moderate enlargement of the spleen have been found should be included in this group, and these constitute the basis for the view now seldom expressed, that splenic anæmia and progressive pernicious anæmia are closely allied. As a matter of fact, in the conditions nowadays classified as splenic anæmia impoverishment of the blood is rarely a predominating feature except after repeated hemorrhages.

2. *Pseudoleukæmia.* The earliest writers (Griesinger, H. C. Wood) suggested that the condition is usually a splenic variety of Hodgkin's disease, and this view is occasionally expressed at the present day. This opinion was adopted by Sippy in his admirable review of the subject, but is now generally abandoned for the reason that the pathological anatomy as far as known almost invariably

negatives the supposed pseudoleukæmic nature of the disease. Nevertheless there are some cases reported in older (Müller) and more recent literature in which the symptoms and such pathological data as were obtained show that splenic pseudoleukæmia may be, clinically, nearly if not quite identical with that we now call splenic anæmia. Such cases are, however, exceptional.

3. *Leukæmia.* All systematic writers call attention to the occasional subsidence of leukocytosis in this disease. Under these circumstances the case might for a time have many of the clinical features of splenic anæmia, and some authorities (Griesinger) regarded the latter as an aleukæmic variety of splenic leukæmia.

4. *Cirrhosis of the Liver.* Some degree of splenic enlargement is habitual in cirrhosis of the liver. Formerly this was attributed to congestion of the spleen caused by obstruction of the portal circulation, but more recently the view has gained ground that the splenic condition results from the action of the same irritant as that which causes the cirrhosis (alcohol, etc.). Under these circumstances it might seem natural that certain cases of cirrhosis should present an unusual degree of splenic enlargement. The studies of Banti, showing a frequent association of splenic anæmia and cirrhosis, gave additional color to this view, and seemed to justify the expression *splenomegalic cirrhosis of the liver.* On the other hand, however, the investigations of Banti, Bovaird, and others showed that clinically as well as pathologically the cirrhotic process may be demonstrably secondary to the splenic disease. Under such circumstances the condition is rather one of *splenomegaly with cirrhosis.* The cases in which pronounced splenomegaly is simply an incident in cirrhosis are probably rare as far as accurate studies would indicate.

5. *Hypertrophic Cirrhosis of the Liver* (Hanot's disease). In this condition the long duration of the disease and the considerable degree of splenic enlargement are the basis upon which inclusion under the head of splenic anæmia has sometimes been suggested if not justified. Some of the cases of familiar splenic anæmia in particular may perhaps be more properly classified here.

6. *Chronic Splenitis.* The greatest confusion exists in the case of splenic enlargement in children. Such enlargement is common in rickets and congenital syphilis, and occurs in the uncertainly classified condition called anæmia infantum pseudoleukæmica (von Jaksch). In the case of rickets and congenital syphilis the splenic lesion is a chronic splenitis leading to fibroid change and some lymphadenoid hypertrophy. Clinically the dominating symptoms are those of the underlying affection. It is, of course, possible that exceptionally the splenic enlargement and its consequences might overshadow other features, but the evidence at hand does not bear out such an assumption. In the case of von Jaksch's disease, path-

ological studies and accurate clinical observations also are still wanting.

The chronic splenitis of malaria belongs in the same group. How far malaria may prove to be a factor in the etiology of splenic anæmia remains unsettled. Undoubtedly, however, there is some added element that operates in the development of the condition even if malaria is a factor.

Leaving out of consideration the cases of splenic enlargement, which by their clinical features or pathological lesions could be plainly referred to the above groups, there remain certain cases in which the splenic disease is of such dominating character that the cases must be regarded as forming a separate group or separate groups. The fact that cirrhosis of the liver supervenes in the late stages does not militate against such a classification, since very good evidence has been brought forward to show that splenic disease may occasion secondary cirrhosis of the liver. Conspicuous predominance of splenic involvement from the beginning warrants the separate classification of such cases even when some evidence of cirrhosis of the liver is an early feature.

It would be hopeless in the present state of knowledge to attempt a classification of all of the cases that have been reported. On the other hand, it is very evident that there are very different sorts of cases that have more or less superficial resemblance, and I cannot share the view that all are probably instances of one disease in different stages. The ultimate solution of the matter must, seemingly, come from more careful clinical and pathological study of these cases, with recognition of the fact that the long continuance of the disease may cause very different lesions at different stages. The following varieties may be recognized provisionally:

1. *Simple Splenomegaly.* In a certain number of cases, including some described by Müller, Banti, Osler, Rendu, Peter, Sippy, and others, the conspicuous feature in the clinical history is the splenic enlargement. Anæmia is not by any means an invariable symptom, though usually present, especially when hemorrhages occur. The hemorrhages may be a purely splenic feature in the sense that they are caused by mechanical obstructions brought about by traction on the splenic vein, or they may be caused by general conditions less readily explained, as, for example, in the case of hemorrhages from the gums. Pathologically speaking, this variety belongs in the category of chronic splenitis such as occurs secondary to malaria, syphilis, rickets, and chronic gastrointestinal diseases. The lesion is essentially a fibrous hyperplasia. In some instances the endothelial cells of the sinuses are swollen and large macrophages are found; in others these cells are not observed, possibly on account of the late stage at which the disease has terminated. Analogy suggests that some form of chronic infection or intoxication is the

cause of this variety. The tendency to some endothelial proliferation does not exclude this view, for in all kinds of chronic splenitis such endothelial processes may occur. In some cases the disease may persist for a long time and the patient may ultimately die without additional lesions; in other cases changes in the liver develop as a final stage and cause a marked alteration in the clinical features. These latter cases are, I believe, in some sort different from the former in their pathology, and may for convenience be separately classed as

2. *Simple Splenomegaly Terminating in Cirrhosis of the Liver.* Perhaps the cases which ultimately develop cirrhosis of the liver and assume the fully developed character of Banti's disease are cases which from the start had a prominent grade of endothelial proliferation in the splenic sinuses. Banti has shown that there may be an endophlebitis extending from the spleen to the splenic vein and eventually to the portal vein, and he referred the cirrhosis of the liver to this cause. In the cases recently reported by Dock and Warthin, stenosis of the portal vein was discovered, and this may have been preceded by a similar endothelial process. In one of my own cases there were, within the terminals of the portal vein in the liver, large endothelial cells corresponding to those found in the splenic sinuses and in the regional lymphatic glands. In Bovaird's case large endothelial cells corresponding with those found in the sinuses of the spleen were a conspicuous feature in the portal spaces of the liver. The probable embolical origin of these formations could not be overlooked. All of these facts suggest that cirrhosis of the liver as a termination of splenomegaly is particularly likely to occur when the process from the beginning has been inclined to affect the endothelial cells in the splenic sinuses particularly. A review of the cases reported in the literature discloses the varying degree of endothelial proliferation that may be present, and it seems likely to me that this is not the result merely of the different stages at which the examinations happened to be made, but rather the consequence of natural difference in the pathology of different cases. Even accepting this, however, the difference may be only one of degree.

That splenomegaly may actually precede cirrhosis of the liver, or at least any considerable degree of cirrhosis, was shown distinctly by one of my cases (Case V.) here reported, in which the patient had been under my observation for over a year preceding the final development of the well-known symptoms of Banti's disease. When she came under observation there was a decided enlargement of the spleen, but no other signs of illness.

3. *Splenomegaly with Marked Constitutional Disturbances.* A certain number of cases of splenomegaly have been described in which, in addition to the enlargement of the spleen, there has been a

profound disturbance of the general health, causing a stunted growth, clubbed fingers, osseous changes, and pigmentation of the skin. Gilbert and Fournier report a group of such cases in children in whom the symptoms were those enumerated, together with enlargement of the liver and jaundice. Frederick Taylor has also reported a series and refers to the case of H. R. Smith, another presented in the Medical Society of the Hospitals of Paris, and some other less certain instances. In some particulars the group of family splenomegaly described by Brill is analogous, though in other particulars more suggestive of the variety of simple splenomegaly. In considering these cases, which have for the most part been observed in young persons or children, the difficulty of distinguishing the disease from hypertrophic cirrhosis of the liver (Hanot's disease) must be kept in mind. Furthermore, pigmentation of the skin of non-icteric character may be accounted for in various ways, and in some of the reported instances the possibility of its being arsenical must be remembered, while in other cases the whole condition may belong to that uncertainly defined affection called hæmochromatosis, in which cirrhosis of the liver is often an incident. Leaving out uncertain cases, however, there seems to be a group or variety in which splenomegaly is associated with stunted growth, pigmentation of the skin, clubbing of the fingers, and other evidences of general systemic disturbances. In this group probably belong two of the cases here reported. These presented very different clinical features from those shown by other instances of so-called splenic anæmia or splenomegaly.

4. *Splenomegaly Primitive.* While it is not possible perhaps to distinguish this variety clinically, there is a sharp distinction in the pathological features. It will be recalled that the essential characteristic of this condition is the presence of irregular oval spaces surrounded with fibrous tissue, and more or less filled with large endothelial cells containing one or more nuclei. All writers agree that these alveoli are formed by the proliferation of the endothelial lining of the splenic sinuses, with fibrous hyperplasia of the surrounding tissue, causing the formation of a coarse reticulum.

Bovaird has suggested that the proliferated endothelial cells eventually form fibrous tissue, and Rolleston and Osler are disposed to admit this as a probability. Harris and Herzog dissent from this opinion. When the descriptions and figures of the cases of Gaucher, Picou and Ramond, Collier and Bovaird are examined, it may not seem impossible that the endothelial process might be the forerunner of an ordinary fibrous change; that, in other words, as Rolleston states, the endothelial proliferation may be replaced by a fibrous tissue process. When, however, the macroscopic as well as the histological features in my own case are considered, it will be seen that the transformation into the ordinary characters

of a fibrous splenitis could not have occurred after a lapse of any amount of time. From every point of view the case suggests a neoplasm. In reporting his case Bovaird considered the respective merits of the neoplasm and hyperplasia theories, and concluded that the condition was rather one of endothelial hyperplasia. He referred to the doctrine of Thoma, that a tumor must be an autonomous growth. On the other hand, all pathologists are agreed that there is no sharp dividing line between the processes which may be properly called hyperplastic and those which are strictly speaking neoplastic. In reviewing the cases of splenic anæmia in which the endothelial hyperplasia has been a feature it will be seen that there are all grades of conspicuousness of this part of the pathological processes—from those cases in which the predominating feature is a fibrous hyperplasia, with a certain element of endothelial proliferation superadded, up to those cases like my own (Case III.), in which the macroscopic features of the condition were those of a tumor and the microscopic characters those of an active neoplastic formation, essentially and primarily involving the endothelial cells.

Pathological Anatomy. Compared with the mass of clinical material the pathological data are scant and unsatisfactory. In part this is due to the chronicity of the disease and in part to the fact that the examinations have been made at various stages, when presumably diverse conditions might be expected. The most extensive of the more recent studies are those of Banti. In brief the following notes contain the results of his examinations:

The spleen is much increased, weighing from 1 to 1.5 kilograms. The shape is preserved, the surface smooth and regular. The capsule is thickened and on section the substance is dark brown and hard, showing thickened trabeculæ and whitish nodules which correspond to the Malpighian bodies.

Microscopically the Malpighian bodies were found in various stages of fibrous transformation, the process beginning around the vessel within the body. A certain amount of hyaline transformation was also apparent. The splenic pulp showed no special enlargement of the venous channels. In the earliest stage there may be found in some of the veins a lining of large cells, making the vessel appear almost like an epithelial channel on cross-section. These large cells have a limited amount of protoplasm and a large nucleus of a diameter of from 5 μ to 8 μ. The same kind of cells are found in the reticulum surrounding the veins, and in some of the channels the cells have become loosened from the walls and fill the lumen of the vessel, giving the appearance of a cancerous alveolus. In some spaces actual giant cells and occasionally cells containing normal or degenerated red corpuscles were observed. The reticulum of the pulp was evidently thickened and of hyaline

appearance. This was always a conspicuous feature and distinguished the tissue absolutely from normal spleen. The thickening of the reticulum is in proportion to the duration of the disease, and finally areas of the splenic pulp may become entirely fibrous. In view of the process of fibrous transformation and the multiplication of cells within the veins, Banti proposed the name "fibroadenia." He does not believe there is a new-formation of fibrous tissue in the ordinary sense of the term, but rather an increase of the reticulum from exudation from pre-existing cells.

The liver is reduced in size, granular, and hard. In earlier stages of the disease it may be normal in size and smooth. Microscopically there is found a marked interlobular cirrhosis like that due to alcohol.

Banti calls particular attention to sclerotic endophlebitis of the splenic vein, causing irregular thickening and sometimes even atheromatous and calcareous foci. These conditions also extend into the portal vein, and in the ascitic stage of the disease even the mesenteric veins may be affected, though in less degree than the splenic.

The bone-marrow showed all the characteristics of fetal marrow, but nucleated red corpuscles were present in small numbers.

Bacteriological examination of various tissues and the blood proved negative.

Most of the reports published since Banti's correspond in all essential details.

According to Osler[1] two conditions have been described in the spleen: (1) a fibrosis and hyperplasia with atrophy of the pulp and a hyaline degeneration of the Malpighian bodies, and with this description, according to MacCallum, Osler's cases Nos. 4, 5, and 16 correspond; (2) on the other hand, very remarkable change is found in certain cases which agree in every clinical particular with the definition of the disease as given for ordinary cases. The normal texture of the spleen is replaced by fibrous tissue and large endothelial cells with clear protoplasm containing two or more nuclei and among them giant cells. This was the condition first described by Gaucher as primary endothelioma.

Sippy[2] could find records of but ten post-mortems in which he felt reasonably certain that the cause of death was "splenic pseudoleukæmia." The liver was usually enlarged, the surface smooth, the edges rounded, and no evidence of cirrhosis, though under the microscope slight increase of the interlobular connective tissue may be found. The spleen is always increased in size, and there may be perisplenitis. Its surface is usually smooth, but sometimes peri-

[1] Transactions of the Association of American Physicians, 1902, p. 429.
[2] The American Journal of the Medical Sciences, November, 1899.

toneal adhesions have been recorded. In a case reported by Banti there was visible increase in the connective tissue, and small white areas about the size of a pea were noted, and these proved to be made up of connective tissue. In Sippy's[2] case there was no macroscopic evidence of connective tissue, but microscopically considerable fibrosis was the feature of interest. Banti summarizes the findings in the spleen as follows: (1) atrophy and sclerosis of the Malpighian corpuscles, and (2) substitution of a fibrous reticulum for the delicate reticulum of the splenic pulp. As a rule, according to Sippy, the Malpighian bodies have been found but slightly altered. In his own case a few were sclerosed, the greater number showing no change.

The following is quoted from the description of West ("A Case of Splenic Anæmia, Splenomegalie Primitive," *Medico-Chirurgical Transactions*, 1896): "The spleen showed slight increase in the trabecular tissue—*i. e.*, slight fibrosis. Malpighian corpuscles are much diminished in size and badly developed. Iron reaction negative." Kanthack examined the tissues in West's case and referred to the presence of giant cells in addition to the fibrosis.

Sippy does not quote extensively from others regarding the pathological anatomy, leaving the impression that the histology as observed by others was practically the same as given in the above.

Dock and Warthin[1] report two cases with anatomical studies. In the first the liver was small and showed a moderate increase in connective tissue, especially in one area. The spleen had a thickened capsule and showed a dense fibrous network enclosing enlarged blood spaces. These were lined and in many cases partly filled with large endothelial or reticular cells containing one or more lightly staining nuclei, and in the majority of cases nucleoli. "The fibrous tissue represents a hyperplasia of the reticulum of the pulp, the endothelial cells lining the blood spaces apparently taking part in the formation of the new fibrous tissue."

The bone-marrow was red and contained an increased number of normoblasts and very few giant cells. The retroperitoneal glands were enlarged and of the type known as hæmal gland.

In their second case the liver was small, adherent to the diaphragm by tough adhesions, and the surface on section was granular. It was the picture of an atrophic cirrhosis on microscopic examination.

The spleen showed marked fibrosis with hyaline change of the new-formed connective tissue. This process was uniform throughout the organ. The blood spaces were lined by large hypertrophic endothelial cells. A few giant cells were found in the blood spaces,

[1] Transactions of the Association of American Physicians, 1903.

and the author suggests that these may have been embolic and of bone-marrow origin.

In both of the cases there was a stenosis of the portal vein, and as the pathological lesions are entirely compatible with chronic congestion of the spleen they suggest that the condition of the portal vein may have been the cause of the lesion.

They find nothing in recent accounts of splenic anæmia suggesting the occurrence of this condition of the portal vein, but several cases were reported by Sir Andrew Clark.[1] The lesions of the splenic and portal vein described by Banti differed somewhat from that reported by Dock and Warthin, though perhaps later stages of the conditions observed by Banti might correspond.

Williamson[2] describes a case which came to autopsy and in which the histological features were sclerosis of the Malpighian bodies, general fibrous hyperplasia, great congestion of the pulp, and sinuses filled with large corpuscles containing cells. The last-named cells contained from six to ten red corpuscles and were mononuclear. I find no other recorded case with such conspicuous macrophages. Allusion is made by some authors to occasional large endothelial cells containing red corpuscles or fragments of such, but not to the numbers found by Williamson nor to cells of the peculiar character figured by him. In a case studied by Rolleston, the spleen, weighing thirty-seven ounces, "showed fibrosis with widespread proliferation of the endothelial cells and disappearance of the leukocytic elements."

Taylor, in referring to one of his cases of "splenomegalic cirrhosis of the liver," states that "the spleen was loaded with red corpuscles; otherwise it appeared normal."

One cannot read the accounts of pathological examinations, scanty and unsatisfactory as they are, without receiving the impression that they very probably refer to different pathological conditions, however similar the clinical features. There are three sorts of changes that may be clearly distinguished: (1) congestion, (2) fibrous transformation, and (3) endothelial proliferation. The first of these was conspicuous in only a few of the cases, and is more prominent in instances (like that of Strümpell) of doubtful character. Fibrous hyperplasia of the splenic reticulum and sclerosis of the Malpighian bodies constitute a marked condition in nearly all of the cases—a fact which is, of course, easily explained by the chronicity of the disease and the late stage at which the histology has usually been studied. What the conditions might have been at earlier stages cannot be determined by such examinations. The endothelial proliferation is not

[1] Transactions of the London Pathological Society, 1867.

[2] Medical Chronicle, May, 1893.

peculiar to cases of so-called splenic anæmia. A certain amount of such proliferation is found in cases of infectious splenitis of all kinds, and this doubtless contributes, in such cases as in splenic anæmia (Bovaird, Rolleston, Osler, Dock and Warthin), to the fibrous change in the reticulum.

A very different condition is that first described by Weichselbaum as "Primary Multiple Endothelial Sarcoma of the Spleen," and later by Gaucher as "Epithelioma primitif," and by Debove and Bruhl as "Splenomegalie primitive." Among the cases of this description are those of Weichselbaum, Gaucher, Collier, Picou and Ramond, Bovaird, and my own (Case III. of this series). The case of Weichselbaum and mine differ from the others in having a more marked neoplastic appearance on macroscopic examination. The opinion of Birch-Hirschfeld that Weichselbaum's case was one of endothelial hyperplasia rather than a tumor does not accord entirely with the gross appearance of the organ, though it is more applicable to Weichselbaum's case than to my own. This will appear from the recorded description.

Weichselbaum[1] remarks, by way of preface, that he was unable to find any instance of primary sarcoma of the spleen in the literature. The specimen was obtained from the body of a soldier twenty-one years old. No clinical data are given. The spleen was "somewhat enlarged," hard, dark brown in color, and presented visible follicles. On section the surface was found studded with small nodules the size of a millet-seed or pea, grayish-red in color, rather soft, and projecting above the cut surface. Their color distinguished them sharply from the splenic tissue, but there was no other sharp demarcation. On microscopic examination there was found a fibrous reticulum, coarser than that of the normal reticulum of the spleen, but no distinct alveolar arrangement. In the meshes of the reticulum were found large endothelial cells of round, oval, or irregular shape, some of the latter having projecting processes. The nuclei were large and often double. In the periphery of the masses large cells containing remnants of red corpuscles or yellowish pigment were found. In the same region extension of the processes of the cells into the fibrous reticulum was observed. All other organs were uninvolved.

In Collier's case the involvement of the spleen was in the form of a zone of cortical infiltration, and Bovaird describes pyramidal formations of white or yellowish-white color extending deeply into the substance of the organ, and involving the greater part of its anterior half, while the posterior half was comparatively free. In Picou and Ramond's case the macroscopic appearance of the organ is not described with sufficient accuracy. In none of these cases

[1] Virchow's Archiv, 1881, vol. lxxxv. p. 563.

was the condition so similar to that of definite neoplasms as in mine. It is not impossible though difficult to conceive how in any of these cases a longer duration could have led to an effacement of the conspicuous cellular process, and a conversion by fibrous hyperplasia into an organ such as those previously described in cases of ordinary splenic anæmia. It may be admitted as probable that endothelial proliferation is a precursor of some of the fibrous-tissue formation in splenic anæmia as well as in chronic splenitis, just as the same process may lead to sclerosis in the peritoneum; but it seems to me just as unlikely that the endothelial process in the six cases under consideration could have terminated in complete fibrosis as that endotheliomata of the peritoneum may have a like conclusion.

It is always difficult to draw sharp lines between cellular hyperplasias and tumors. In the case of glandular affections of the gastro-intestinal mucosa, in certain epithelial proliferations in the liver, thyroid gland, and breast, this difficulty is well recognized. The microscopic features alone do not suffice, and an appeal must be made to the gross appearances, the etiological relations, etc. Viewed in this way, I cannot avoid the conclusion that my own case belongs in the category of tumors rather than of simple cellular hyperplasia. The peculiar prominence of giant cells naturally raised the suspicion that the process might be infectious or parasitic, but no form of bacterial organisms nor anything suggesting protozoa could be discovered.

One of the arguments advanced in opposition to the tumor theory in such cases is the preservation of normal splenic appearances. As one author states, "After all these years the organ is still a spleen." This preservation of normal appearance is, however, more superficial than real, as sections through the organ showed in several cases almost complete transformation of the splenic substance.

The conclusion to which I have come is that the cases of splenomegaly of the type of Gaucher represent a primary neoplasm of the spleen somewhat comparable to the diffuse myelomata, and to certain infiltrating sarcomata of the liver.

REVIEWS.

CLINICAL TREATISES ON THE PATHOLOGY AND THERAPY OF DISORDERS OF THE METABOLISM AND NUTRITION. Part IV. The Acid Autointoxications. Part V. Concerning the Effect of Saline Waters on Metabolism." By Prof. CARL VON NOORDEN and Dr. CARL DAPPER. New York: E. B. Treat & Co., 1904.

MOST interestingly expressed and clearly defined are the authors' views on the difficult subject of defective metabolic processes with the resultant intoxications. Acetone, formerly regarded as the cause of diabetic coma, is proven to be the least significant of the three acetone bodies: oxybutyric acid, diacetic acid, and acetone. The acetone bodies are regarded by the author as products which, under normal conditions, should have undergone further oxidation, that is, that with proper supply of oxygen they would not exist in the urine at all except as acetone in very small quantity. The appearance of β-oxybutyric acid in the urine, the undoubted cause of diabetic coma, is indicative of a very grave defect in oxidative function. The fact that the acetone bodies are non-toxic when taken by the mouth leads to the view that their effects are produced by union with the fixed alkalies of the body, and are thus poisonous because acid. Acetonuria, having been produced experimentally by starvation as well as by persistence in a meat-fat diet, and having been made to disappear by the addition of carbohydrates to the diet, the author is led to the conclusion that the acetone bodies appear solely because of lessened carbohydrate feeding. In regard to the *modus operandi* of the carbohydrates in this connection the suggestion is advanced that the influence is a contact effect, oxygen being liberated within the cell. In diabetes where the acetone bodies are excreted in quantities greater than in any other condition the cause is traced to several sources: the destruction of those cells which should aid in the disassimilation and oxidation of the bodies, the failure of combustion of even the small amounts of carbohydrate administered, and finally the small quantity of carbohydrate food actually taken. β-oxybutyric acid and diacetic acid may be made to disappear altogether in some cases of diabetes by administering certain quantities of carbohydrates, but this effect can never be produced unless some quantity of the carbohydrates undergo combustion and not all be passed unused. In such cases no benefit, but in all probability positive harm, would result from the administration of carbohydrates. von Noorden frankly points

out several apparent fallacies in this theory of the etiology of the appearance of the acetone bodies, but as the fallacies are not insuperable we can afford to accept the theories and look for further elucidation along the same lines. The therapeutic measures of combating acidosis based on the theories evolved are divided into (1) means directed to the limitation of the formation of the acetone bodies; (2) means directed toward the disintoxication of the bodies circulating through the tissues and toward accelerating their elimination. The first is employed with every hope of immediate success in non-diabetic cases. In diabetes the difficulties are much greater, and here the treatment consists in careful administration of carbohydrates until the acidosis is diminished in quantity or that happy result obtained, the disappearance of all acid. von Noorden has had great success follow his oat-meal treatment. Finally, we have the power in many cases of neutralizing the acid by free use of sodium bicarbonate and sodium citrate given daily by mouth, or intravenously as sodium carbonate, in diabetic coma.

Part V. of von Noorden's series of treatises deals with the action of saline waters on metabolism, especially in those conditions most frequently seen at watering-places. While all the patients under the care of the author are put upon rigid measured diets, yet that this alone is not responsible for the cures reported is satisfactorily shown by control experiments in which the diet was not assisted by the waters, with, in most instances, less satisfactory and less rapid improvement. The effect of the saline waters is discussed in cases of alcoholic dyspepsia, hyperacidity, subacidity, anacidity, and obesity, and its use in aiding the elimination of uric acid. The author believes that the exclusion of fruit, vinegar, salads, etc., from the diet is not based on findings of scientifically conducted experiments. There are many exceptions taken to old fetishes held sacred at certain spas. von Noorden finds that fat in large quantities is readily absorbed by patients partaking of alkaline waters, and in selected cases is of the greatest benefit in the treatment, while, on the other hand, in treating obesity where fat in the diet is restricted or altogether interdicted, the waters do not increase the proteid metabolism, and thereby weaken the patient, as has been claimed. The treatise is full of interesting information conveyed in an authoritative way. J. N. H.

EVOLUTION AND ADAPTATION. By THOMAS HUNT MORGAN, Ph.D. New York: The MacMillan Company, 1903.

THIS book, which numbers 464 pages, is arranged in the form of a critical review of the various theories and hypotheses which have been advanced for and against the doctrines of evolution and

adaptation. The subjects are treated in a scientific but free manner. Darwin's *Origin of Species* is extensively quoted from and discussed at length.

After analyzing the views brought forward by other workers, the author draws certain conclusions, which differ in many points from those of Darwin and others, especially as regards the origin of species and the importance of adaptation to environment in the determination of these species.

The book is well printed on good paper and forms altogether an interesting treatise. W. T. L.

AN INTRODUCTION TO VERTEBRATE EMBRYOLOGY, BASED ON THE STUDY OF THE FROG AND THE CHICK. By ALBERT MORSE REESE, Ph.D. With 84 illustrations. New York and London: G. P. Putnam's Sons. The Knickerbocker Press, 1904.

THIS small book presents the subject of embryology in a concise and understandable form, and, as an introduction to the study, is especially adapted to students. The development of the frog and of the chick are separately considered. The first chapter, a rather long one, follows the series of change in the growth of the frog from the ovum to the adult, while the remaining seven chapters deal with the development of the chick. In considering the development of the chick a more or less arbitrary but useful division is made, inasmuch as the stages are described in periods, the first five days taking up five chapters.

The larger treatises and text-books upon the subject have been freely consulted. The illustrations are numerous and fairly satisfactory, being adapted from other publications. As an outline for the study of embryology the book seems very valuable.

W. T. L.

TRAITE DE HEMATOLOGIE. PAR FERNAND BEZANCON ET MARCEL LABBE. Avec 125 Figures et 9 Planches en couleurs, d'apres les Aquarelles de M. Labbe. G. STEINHEIL, editeur. Paris, 1904.

THE recent studies upon leukocytogenesis and upon the origin in general of the formed elements of the blood have, among other things, emphasized the close connection which exists between the hæmopoietic organs of the cells in the circulating blood. For a proper understanding, therefore, of the alterations in the blood cells during pathological conditions some knowledge of the condition of the hæmopoietic organs must be gained, since the changes in these organs and those in the circulating blood are, after a manner, interdependent. In the present volume advantage has been taken of this fact, and a rather unusual treatment of the subject

of hæmatology produces a result that is both interesting and refreshing. The work, which is quite large, covering over 900 pages, cannot be considered in the light of a text-book for students. The technique of blood examinations is relegated to a position of minor importance, and, instead of the usual concise and hackneyed descriptions of the blood in various conditions, each element of the blood is considered separately in its relation to various systemic and local diseases, and also in its relation to the hæmopoietic organs. The book is divided into several parts. The first part deals with the general properties of the circulating blood, the bacterial and chemical analysis, while the second part takes up the technique of blood examinations. After this introduction the red blood corpuscle is considered, including a short chapter on hæmolysis. A fourth division takes up the white blood corpuscle and a fifth the hæmatoblasts. The various properties of the serum, including a chapter upon bacteriolysins, cytolysins, agglutinins, and precipitins, make up the sixth part; the seventh and last part is principally devoted to a description of the hæmopoietic organs. The beauty and excellence of the colored plates are especially to be noted.

It is to be regretted that important phases of anæmia and leukæmia are considered in rather a meagre manner compared to the elaborate treatment of the other subjects.

References to original articles are numerous, and the various sides of disputed questions are fairly well presented. In this way a vast amount of valuable data has been collected, and what is not absolutely new often seems so from the manner in which it is considered. W. T. L.

RABIES: ITS PLACE AMONG GERM DISEASES AND ITS ORIGIN IN THE ANIMAL KINGDOM. By DAVID SIME, M.D. Cambridge: The University Press, 1903.

AFTER reading Dr. Sime's book one is at a loss to know why it was written or what purpose it serves. Throughout the various chapters the author unfolds and elaborates certain hypotheses concerning the mode of transition of the disease, the character of the rabies virus, and the structure of the rabies germ, the incubation period of the disease, and the changes in preventive inoculation. The work is largely based upon the author's own interpretation of Pasteur's experiments, which are extensively quoted and turned to suit the occasion. There is profound disregard for, or perhaps ignorance of, the modern conceptions of bacteriology, while the recent investigations upon rabies receive no mention whatsoever. The greater part of the book deals with the rabies-germ, and though the author admits on page 86 that "as yet the rabies microorganism itself has not been conclusively identified," this fact does

not deter him from picturing it in its many forms as he conceives it. Further, the book is teeming with inaccuracies and misstatements, as, for instance, on page 99, where, in speaking of the "prophylactic or non-recurrent" infections, and the "preventive or recurrent" infections, the following statement is made: "Under the first head would be comprised such *germ diseases* as smallpox, anthrax, syphilis, scarlatina, measles, whooping-cough," etc. Again one finds on page 103: "There can be no doubt that like anthrax, smallpox, syphilis, and the entire prophylactic order it (rabies) affects animals in common with man, and particularly the domestic animals." From certain foot-notes it seems possible that the book was really written several years ago. This would probably be the kindest explanation for its existence. W. T. L.

STUDIES IN HETEROGENESIS. By H. CHARLTON BASTIAN, M.A., M.D. Lond., F. R. S. With 815 illustrations and photomicrographs. London: Williams & Norgate, 1903.

IN the preface the author states that his observations admit of three interpretations: (1) "It may be said that the forms of life here described, which seem to take origin from substance of other organisms or their germs, are not alien organisms, but normal stages in the life-history of the present forms." (2) "That the resulting forms of life are due to the invasion and multiplication of parasites within what appear to be the present organisms." (3) "That the resulting forms of life are, in reality, heterogenetic products originating from the very substance of the organisms or of the germs from which they proceed."

The third interpretation is chosen, and the term heterogenesis is considered to mean "processes by which living things arise from the matter of pre-existing organisms belonging to a totally different species."

The entire book, numbering almost 400 pages, resolves itself practically into a compilation of the record of observations upon what seems to the author to be a transformation of certain lower forms of life from one species to another. But, aside from the biological questions involved, the technique of most of the experiments is not such that one would be disposed to consider the results in a very serious light.

Indeed, when one reads a chapter in the appendix "On the Great Importance from the Point of View of Medical Science of the Proof that Bacteria and their Allies are Capable of Arising *de novo*," he feels much as if he were dealing with a pre-Pasteurian treatise. W. T. L.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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On the Occurrence of Lipase in the Urine as a Result of Experimental Pancreatic Disease.—In pancreatic diseases in which fat necroses occur it is generally believed that the latter are due to the action of lipase, the fat-splitting ferment of the pancreatic secretion. It is probable that the ferment reaches the fat cells by various channels. It may become diffused by an exudate throughout the abdominal cavity; it may travel through the areolar tissue, or it may be carried in the lymph and blood currents. The great preponderance of cases in which the necroses are practically limited to the immediate neighborhood of the pancreas or to the subperitoneal fat favors the first two methods. The fact that the fat necroses have been found in the pericardial, subpleural, and subcutaneous fat of the chest and abdomen and in the intermuscular tissue and bone-marrow, suggests very strongly that, in exceptional cases, the ferment is carried in the lymph and blood stream to distant parts of the body.

HEWLETT (*Journal of Medical Research*, May, 1904, p. 377) has carried out a series of experiments on dogs with the view of determining the relation, if any, between experimental disease of the pancreas and the presence of a fat-splitting ferment in the urine. Lesions of the pancreas were produced by injecting various substances into the pancreatic duct, ligaturing the duct, etc. Without going into the details of the writer's method of determining the presence of lipase in the urine, the essential features of the test are as follows: Equal quantities of urine are poured into two flasks. The contents of one flask are boiled to destroy any ferment that may be present. Both urines are neutralized with sodium hydrate. A measured quantity of ethyl butyrate and a little toluene are added to each, the latter to prevent bacterial growth, and the flasks placed in a thermostat at 39° C. for twenty-four hours. The contents of each are then extracted with ether, and the relative acidity of the two ether extracts is determined by titrating with $\frac{N}{10}$ potassium hydrate solution. Any decided dif-

ference between the acidity of the ethereal extracts of the boiled and of the unboiled urine is due to the butyric acid formed by the cleavage of the ethyl butyrate by the lipase, and where the difference in acidity is at all marked the odor of the butyric acid can be easily recognized.

Ten dogs were operated on. At the autopsy, performed at varying periods after the operation, fat necroses were found in eight. In all these a fat-splitting ferment was demonstrated in the urine. The experimental pancreatic lesions in which lipase was found in the urine were acute pancreatitis, acute obstruction of the pancreatic duct, and pancreatic trauma. Hewlett expresses the belief that lipase is present in the urine at some time during the course of most pancreatic diseases which give rise to fat necroses. It was found in greatest amount in experimental acute hemorrhagic pancreatitis. It also occurred from three to five days after obstruction of the pancreatic duct. These are the two conditions in which fat necroses are most apt to occur in human pancreatic disease. Hewlett states that very little, if any, lipase is present in normal urine. It appears, therefore, that the finding of lipase in the urine is of considerable diagnostic importance in determining the existence of pancreatic disease.

A Contribution to the Knowledge of the Purin Bodies of Human Feces in Health and Disease.—Walker Hall (*Journal of Pathology and Bacteriology*, March, 1904, p. 246) has conducted a series of investigations to determine the quantitative excretion of purin bodies (uric acid plus the xanthin or purin bases) in the feces, to ascertain the absorption quotients of the several purin bases, and to estimate the output of the fecal purins in morbid conditions.

The purin bodies of the feces may arise from (1) desquamation of the columnar epithelium of the alimentary canal; (2) the nucleoproteids of bacteria and leukocytes; (3) the fluids poured into the intestine by its secreting cells and by those of the pancreas. Bile has been shown not to contain any purin bodies. There is no evidence to indicate that either xanthin or uric acid are excreted from the tissues of the body into the intestines. In a few stray cases uric acid has been found in the feces, but only in minute quantities.

The research seemed to bring out one point which may modify our views regarding the diet of gouty patients. The thymus sweetbread and the pancreas sweetbread are both known to be rich in purins, the former in adenin and the latter also in adenin, and, to a larger extent, in guanin. Both these glands have been considered injurious for the gouty individual, owing to their richness in these purin or nuclein derivatives. Hall's investigations show that a very large percentage of the adenin of the thymus is not absorbed and is rapidly excreted, and that the guanin of the pancreas is incapable of increasing the urinary purin output and of exerting any injurious effects on the tissues. This is a return to the position of Luff, who, as a result of clinical experience, recommended the use of sweetbread in gout.

Walker Hall finds that the fecal purins show an average daily excretion in the same individual at different intervals and under similar circumstances, the quantity ranging from 0.01 to 0.03 gm. of purin nitrogen on a mixed diet. The addition of medium quantities of meat and hypoxanthin do not alter the normal amount, and they are, therefore, probably fully absorbed. When excessive amounts of meat,

thymus glands, or guanin are added to a mixed diet a large proportion of their purin nitrogen is excreted in the feces within the following twenty-four hours. The increase is due to an increase in the guanin and adenin constituents respectively. The xanthins are slightly increased, but this is probably due to oxidation processes. The fecal purins are increased in diarrhœa. He states that the prescribing of sweetbreads to patients, the subjects of metabolic disorders, seems justified by the results of his investigations, which, however, indicate that foods containing hypoxanthin and xanthin should be limited.

Walker Hall's investigation is of much interest and importance to those carrying on metabolism investigations.

SURGERY.

UNDER THE CHARGE OF

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Rhythmical Traction of the Neck for Respiratory Syncope Due to Anæsthesia.—DEPAGE (*Jour. de Chir. et Annales de la Soc. Belge de Chir.*, January, 1904) states that the rhythmical tractions of the neck have exactly the same effect as the rhythmical tractions of the tongue. The manœuvre consists in standing behind the head of the patient and then stretching the neck backward and forward twenty or thirty times a minute. This method is to be preferred to traction of the tongue for the following reasons: 1. It is easier. 2. It requires no special apparatus, such as mouth-gag or tongue-forceps. 3. It does not cause a wound. 4. It may be done by the anæsthetist without assistance by the operator.

The Leukocyte Count in Appendicitis.—HEAD (*Northwestern Lancet*, February 15, 1904) states that in a recent article in the *Münchener med. Woch.*, Rehn observes after a study of 19 cases that this procedure is of no value in the diagnosis nor prognosis of the disease. Head, after a careful study of 30 cases, states that these conclusions are too sweeping and do not express the truth of the matter, and the following conclusions are more nearly correct: The white blood count will *not* be of service in determining (*a*) whether perforation is or is not present; (*b*) whether perforation is about to take place; (*c*) whether the appendix is gangrenous or not; (*d*) whether peritonitis complicates the appendicitis; (*e*) whether the patient will or will not recover without operation.

The white blood count will determine (*a*) whether the appendicitis

is catarrhal or suppurative in the majority of cases; (*b*) whether the inflammatory process is spreading or not; (*c*) whether the disease is appendicitis or typhoid fever with appendical symptoms; (*d*) whether the disease is appendicitis or acute abdominal pain from renal calculus, gallstones, gastric ulcer, acute nephritis, acute obstruction, etc.

The laboratory procedures should be regarded as merely aids to diagnosis and not as infallible evidence for or against a certain diagnosis. They should never be studied apart from the symptoms presented by the patient and often require a special interpretation by someone familiar with the modifications of the test produced, oftentimes by the same disease at its various stages and under varied conditions.

Atrophic Cirrhosis of the Liver, Ascites, Omentofixation; Recovery after Two Years.—JONNESCO (*Bull. et Mém. de la Soc. de Chir. de Bucarest*, 1904, No. 6) reports the case of a man, aged sixty-eight years, whose condition was most grave at the time of operation, but who now, two years later, has had no return of the ascites and is apparently well. The operative procedure consisted in a double omentofixation, one intraperitoneal and the other interhepato-diaphragmatic. In order to do this the omentum was separated into two halves, one half was fixed in the sheath of the muscle on the right side, while the other was fixed by sutures against the convex face of the liver, which had previously been scarified, and the diaphragm.

Two Cases of Cysts of the Knee.—PATEL and VIANNAY (*Revue d'orthopédie*, 1903, No. 4), after reporting two cases in detail, state, in conclusion, that there exists on the internal aspect of the knee, in the region of the internal tuberosity of the tibia, between the internal surface of the superficial fascia and the external surface of the aponeurosis, a space which in four out of every fifteen cases is a true serous bursa analogous to the prepatellar bursa. There may exist isolated cysts developed in the bursa of the tendon of the semimembranosus and tending to invade the common bursa. On the internal surface of the knee, in the region of the tuberosity of the tibia, between the cellular subcutaneous tissues and the aponeurosis of the leg, one finds occasionally a serous bursa which may become enlarged as the result of traumatism.

THERAPEUTICS.

UNDER THE CHARGE OF

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Yersin's Serum in Bubonic Plague.—DR. LOUIS CAIRUS, after an experience with this serum, states that in it we have an agent of great therapeutic value in the treatment of bubonic plague; that its action is not only bactericidal, but antitoxic as well; that this double action is most efficacious when it is administered early in the disease, and is

injected both intravenously and into the substance of the buboes; that in light cases, perhaps, subcutaneous injection alone is sufficient, but in severe infections the combined method is necessary. In these latter the beginning dosage should be from four and a half to nine ounces, the intravenous dosage to be regulated according to the severity of the constitutional symptoms.—*Therapeutische Monatshefte*, 1904, No. 5, p. 221.

Antistreptococcic Serum in Acute and Chronic Rheumatism.—M. MENZER has treated with his antistreptococcic serum forty-seven patients affected with acute articular rheumatism, all of whom recovered without passing into the chronic stage. From one to two years have passed since the treatment, and no recurrences have taken place. In nine patients who had gone on to the chronic stage of the disease the treatment succeeded completely in seven, while considerable amelioration took place in the other two. In eight of these cases the results achieved have been permanent. In two other analogous cases, complicated by polyneuritis, one cure and one amelioration have taken place. Of eleven chronic cases five were cured, two much improved, and four were unaffected. Of the seven in which favorable results were achieved, five have been followed, and there has been no relapse. Of thirty-four subjects affected for the first time with acute rheumatism and treated by the serum, only one has a valvular heart lesion, and of the total number of forty-seven cases treated in only two has cardiac involvement been observed.—*La Semaine Médicale*, 1904, No. 18, p. 142.

A New Antidysenteric Serum.—DR. ROSENTHAL has elaborated a serum, by immunizing horses against the toxin of dysentery, which he used during an epidemic in the fall of 1903. One hundred and fifty-seven cases were treated, in forty of which the bacillus of dysentery was sought and found. Each patient received from five drachms to four and one-half ounces of the serum subcutaneously. The injection was painless, but in 13 per cent. of the cases was followed by an erythema; in four cases by urticaria; by arthralgia in two cases. There were eight deaths, three of which were due to other infections. All the patients entered the hospital after having been ill for a week or less, and were in the later stages of the disease. The mortality was 4½ per cent., as against from 12 to 17 per cent. in cases not treated by the serum. The serum caused all the symptoms to improve; within twenty-four hours the tenesmus diminished, the stools became fewer, and the blood disappeared. Rarely did a patient go on to the chronic stage of the infection. When the serum was used within three days of the inception of the disease recovery was established in from one to three days.—*Deutsche medizinische Wochenschrift*, 1904, No. 19, p. 691.

The Roentgen Rays in Splenic Leukæmia.—M. M. GUILLOZ and SPILLMAN have treated a girl, aged seventeen years, afflicted with disease, who had shown symptoms for two years. The rays were applied only to the splenic area, and the treatments were given for five minutes every three or four days. After each series of four or five applications a diminution of the leukocytes and of the volume of the spleen was

noticed, and the patient's general condition improved. Epistaxis, which had been a feature of the case, ceased. From the results achieved it may be observed that the Roentgen rays are able to affect deep tissues without causing superficial lesions, and that this action may be utilized to diminish the number of white blood cells in cases of leukæmia.—*La Semaine Médicale*, 1904, No. 22, p. 174.

Red Light in Noma.—Dr. W. O. Motschan reports a case of this serious affection in a boy aged nine years. The inflammation had gone on to perforation of the cheek and lip before treatment was begun, but after three days of exposure to the light of a sixteen candle-power incandescent lamp with red glass and fitted with a conical reflector, the pain ceased and the patient could freely open his mouth. Removing the lamp caused a return of the pain. Soon the secretion of the ulcer became diminished, and healthy granulations appeared upon its floor. The lesion progressively healed, until finally a small sequestrum from the mandible came away, and less than three months after the institution of the treatment there remained only a small rose-colored scar. The distance of the lamp from the ulcerating surface was about eight inches, and the exposure was continuous day and night.—*Wiener klinisch therapeutische Wochenschrift*, 1904, Nos. 21 und 22, pp. 575 and 606.

Injections of Soluble Mercury in Syphilis Neonatorum.—MM. Schwab and Levy-Bing advise this treatment on account of its lack of danger and its rapidity and efficacy. It is applicable in grave cases with visceral involvement. It does not disturb digestion and is free from the inaccuracy of the method by inunctions. The authors use an aqueous solution of the biniodide, the minimum daily dose being 0.015 to 0.03 grain for a child weighing from five to seven pounds. A series of ten or fifteen injections are given, and after an interval of two weeks a second series is administered. The treatment is to be continued at intervals in this manner. The following formula is employed: mercury biniodide, 0.05 part; sodium iodide, 0.05 part; aqua destillata, 10 parts. The solution is injected deeply into the muscular tissue, and is not likely to cause irritation.—*Journal de Médecine de Paris*, 1904, No. 19, p. 186.

Adrenalin in Hemorrhoids.—Dr. M. Demay de Certant recommends as an application to prolapsed hemorrhoids cocaine hydrochlorate, one-twentieth of a grain; adrenalin, 1:1000, thirty drops; water, one ounce. The anal region should be cleansed and a pledget of cotton saturated with the above solution applied and held in place by means of a rubber dressing. The application should be renewed every three hours, until the pain has ceased. Under the influence of the adrenalin the congestion diminishes, the hemorrhage stops, the pain ceases, and the tumors become reducible. In cases of internal hemorrhoids an ointment of cocaine hydrochlorate, one-twentieth of a grain; adrenalin, thirty drops; vaselin, one-half ounce, will be found efficacious. Suppositories of like composition act well, but are likely to cause a painful defecation. While this treatment is slow in action it is sure and lasting in its effects.—*Journal de Médecine de Bordeaux*, 1904, No. 20, p. 352.

The Iodides in Cardiac Conditions.—M. LEFEVRE believes that the iodides are indispensable in organic heart disease, and that their administration should be continued for considerable periods of time in connection with some heart tonic, such as sparteine. The author's observations show that such medication continued for several years may cause the signs of simple endocarditis to disappear. He considers potassium iodide preferable to sodium iodide, since it is more active and less likely to produce toxic effects. The iodide is given twenty days in the month in dosage of five to eight grains daily. During the other ten days one-half to one grain of sparteine is given. This drug is not cumulative, is easily eliminated by the kidneys, does not disturb digestion, and may be continued for a long period after the cardiac symptoms have disappeared.—*Journal de Médecine de Paris*, 1904, No. 20, p. 199.

Diuresis by Reduction of the Ingested Fluids.—MM. HUCHARD and FIESSINGER believe that this procedure is useful in cases of dilated heart where the œdema increases in spite of digitalis and milk diet, as well as in obesity. In the cardiac cases the fluid taken should be reduced to three pints daily—water, one-third; milk, two-thirds—with a glass or two of red wine. Within two days a diuresis is set up, which lasts two, three, or more days, with abundant elimination of the chlorides. This method does not act upon the heart itself, but lessens the work of that organ, and under the reduction of fluids small doses of digitalin, large doses of which had no effect previously, act most favorably. On the second and third days of the reduction four grains of caffeine are given, and the treatment is completed by prescribing a diet of milk and vegetables, from which chlorides are excluded as far as possible, and the administration of seven and one-half grains of theobromine three times a day.—*Journal des Praticiens*, 1904, No. 19, p. 297.

Methods of Using Collargol.—E. DESANTI suggests the following: For inunctions collargol may be mixed with simple cerate in the proportions of 15 to 100, or it may be added to an equal amount of lanolin and mixed with benzoinated lard in the proportion of 30:100. One-half to one drachm may be rubbed into the skin three times a day. A healthy part of the skin should be chosen, and one richly supplied with lymphatics. The part should be cleansed with soap, water, and alcohol, and the inunction should last at least one-half hour—better one hour—and then, in order to insure complete absorption, the skin should be covered with an impermeable dressing. Intravenously or hyperdermically, collargol may be given in 1½ or 2 per cent. aqueous solution, the adult dose being from one-half to two and one-half drachms. Schmidt has injected intravenously one-half to one drachm of a 5 per cent. solution, with no bad results. He uses a hypodermic syringe plunged directly into a superficial vein. In digestive disturbances collargol may be given by mouth in solution or in pill form. Two to five coffeespoonfuls of a solution (collargol, five parts; albumin, one part; water, one hundred parts) may be given. The so-called Credé's pill consists of collargol, fifteen grains; milk-sugar, one hundred and fifty grains; glycerin, q. s. to make one hundred pills; dose, two to six daily. This agent may also be used in surgery as an irrigation

in strength of 1:1000; as a soluble urethral bougie, of 2 per cent. strength, and as a rectal or vaginal suppository, three-quarters of a grain to each suppository.—*Revue Francaise de Médecine et de Chirurgie*, 1904, No. 21, p. 501.

OBSTETRICS.

UNDER THE CHARGE OF

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Puerperal Eclampsia Twice in the Same Patient.—In the *British Medical Journal*, June 25, 1904, McCann reports the case of a patient in her second pregnancy, whom he saw at seven months with highly albuminous urine and beginning labor with partial dilatation. She was soon delivered of a stillborn child. After delivery the patient had a most severe convulsion, from which she seemed to have died. Under vigorous stimulation she gradually rallied, and finally made a good recovery.

The year previously, in her first pregnancy, this patient had eclampsia, and labor was induced at the sixth month. She had prolonged convalescence, with very slow return to health. In the present pregnancy she had been upon a restricted diet and had seemed in good health.

Intrauterine Infection of the Fœtus in Variola.—In the *British Medical Journal*, June 25, 1904, Cowie and Forbes, of the Health Department of Manchester, report three cases in which newborn children were seized with variola so soon after birth that the infection must have been received within the uterus. In spite of immediate vaccination the cases proved fatal. It is estimated that in one case infection occurred two or four days before birth; in another, seven days before birth, and in the third seven days before birth.

The case of a patient in the seventh month of pregnancy attacked by variola is also reported. She was discharged from the hospital a month after admission. A month later her child was born, showed no signs of variola, and was successfully vaccinated. In another case a five-months' fœtus was expelled in the fourth week of the mother's variola, and showed no signs whatever of the infection.

Eclampsia Treated by Thyroid Extract.—Sturmer (*Journal of Obstetrics of the British Empire*, June, 1904) describes 41 cases of eclampsia in the Government Hospital at Madras, India, treated by thyroid extract. These patients were given ten grains of thyroid extract as soon as admitted to the hospital, and five grains every four hours afterward. If the secretion of urine were scanty, saline injections were given, and hypodermic injections of morphine, one-half grain, were

also used if indicated. Under this treatment the mortality was 12.2 per cent. Saline injections did not cause increase in the urine until twenty-four hours after the beginning of treatment, while the hypodermics of morphine caused the child to be somnolent and to breathe badly.

Other treatment, in a series of 369 cases, gave a mortality of 28.7 per cent. The mortality under the use of thyroid extract, morphine, and saline injections was the lowest obtained in this hospital.

Separate Existence in the Child.—ATKINSON (*Journal of Obstetrics of the British Empire*, June, 1904) discusses the question of the exact status of the fœtus as regards its existence as a living being. The separate true existence of the child commences with the isolation of its circulation. This is brought about by the establishment of respiration or by the shrinking of the placental site in the mother's uterus. The child may attain complete independence of the mother before partial birth. There may be a brief intramaternal phase of existence when the child has a separate physiological existence, but is not legally considered a separate individual. The laws of most countries do not recognize infanticide until the child has been expelled from the body of the mother. The death of the fœtus resulting from injury during labor is commonly ascribed to the accidents of labor.

A Case of Quintuplets.—NIJHOFF reports in the *Zentralblatt für Gynäkologie*, 1904, No. 23, a case of the birth of five children. The mother was a healthy woman, aged thirty-four years, who seven years previously had given birth to a healthy male child. She had an abortion six months before the multiple birth. During her pregnancy the patient was well, but the abdomen increased rapidly in size. During a portion of the time there was a discharge of blood and clear serous fluid. In the sixth month of the pregnancy the five fœtuses and placenta were expelled during an hour's time. The first and second children were girls, the third a boy, and the fourth and fifth girls. Before the birth of the first and third children an amniotic sac presented. The first child was born in first position, vertex presentation; the second in footling presentation; the third in second position, vertex presentation; the fourth and fifth in footling presentation, with but partial extension of the feet. The placenta was readily expelled fifteen minutes after the birth of the children by gentle pressure on the fundus uteri. There was considerable uterine hemorrhage during the labor, and clots were passed freely during the puerperal period. Aside from this the patient's recovery was uneventful.

There was but one placenta, divided into three portions of unequal size. The two smaller portions belonged to the third and fourth children each, the septa being composed of amnion and chorion. The remaining greatest portion gave nourishment to the first, second, and fifth children. The septa between these children were composed of amnion only, the umbilical cords anastomosed. The first, second, and fifth children were triplets; the third and fourth were duovular twins.

The mother of the patient had given birth to twins, and other relatives had given birth to twins and triplets. In thirty collected cases it was found that birth usually occurred from the fourth to the sixth

month. The accounts of the placentæ in the different cases vary greatly in clearness and accuracy. In the John Hunter Museum, in London, is preserved a specimen of quintuplet pregnancy, and in the Kennedy Museum, in Dublin, there is a specimen of five children. It is not known that a permanent preparation of the placenta of quintuplet pregnancy is in existence.

Psychosis during Pregnancy Rendering Abortion Necessary.—TREUB (*Zentralblatt für Gynäkologie*, 1904, No. 23) reports the case of a woman, aged thirty-one years, who had had one severe labor followed by normal birth. In her fourth pregnancy she became convinced that the child would be a monster, and she had been greatly frightened by reading a novel which described such a birth. She endeavored to throw herself under a railway train, and tried to kill the monster by introducing a pin through the abdomen. She was admitted to the hospital, and every effort made to overcome the delusion, but without success. It was necessary to empty the uterus, when the patient immediately recovered.

Cæsarean Section in Eclampsia.—In a recent paper before the Edinburgh Obstetrical Society, CROOM (*British Medical Journal*, June 18, 1904) reported two cases in which he had performed Cæsarean section for eclampsia, and reviewed a series of fifty-four cases in the practice of others. The maternal mortality in the series had amounted to about 50 per cent., which was due to the desperate condition of the patients at the time of operation. He believed that the uterus should be emptied in eclampsia as quickly as possible, and that Cæsarean section is the best method of accomplishing this result.

GYNECOLOGY.

UNDER THE CHARGE OF

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Stypticin in Gynecology.—WEISSBART (*Heilkunde*, 1903, No. 10) has met with the best results in the use of this drug in cases of subinvolution. When hemorrhage continues after stypticin has had a fair trial he infers that the uterus must contain placental remains. He would accordingly regard it as a valuable aid in differential diagnosis in cases in which there is no urgent indication for curettement.

Pathogeny of Deciduoma Malignum.—RECASENS (*Revista de med. y cir. pract.; Zentralblatt für Gyn.*, 1904, No. 18) holds that the fetal ectodermal structures possess a specific malignant character, which is usually neutralized by the secretion of the thyroid or ovary(?); when

for any reason the normal secretion of these glands is arrested, abnormal development of the fetal structures occurs.

Case of Ovarian Chorioepithelioma.—JAFFE (*Archiv für Gynäkologie*, Band lxx., Heft 3) reports a case of ovarian tumor which developed two months after the discharge of an hydatid mole. It proved to be a chorioepithelioma. The right ovary, which was as large as an apple, showed the same histological structure. In both tumors there were multiple corpus luteum cysts, a condition which has been noted by many observers to accompany molar pregnancy. The writer believes with Pick, that overproduction of corpus-luteum tissue is the principal feature in these cases, which seems to support Fraenkel's theory that there is a direct causal relation between the two conditions. The excess of corpus luteum material in the blood leads to an increase of trophoblasts, and hence the chorioepithelial development in the uterus.

The Permanent Results of Operations for Prolapsus.—JACOBY (*Ibid.*) reports 257 cases from the Breslau and Asch's private clinics, in 200 of which the patients were kept under observation. In most of these Alexander's operation or vaginofixation was combined with plastic operations. Of the latter class 81 per cent. were cured; of the former, 95 per cent. Vaginal extirpation was performed thirty-five times with 89 per cent. of cures in 27 patients who were examined subsequently.

Prolapsus Uteri in the Newborn.—BURGER (*Zentralblatt für Gynäkologie*, 1904, No. 19) reports the case of an infant who was born with a complete procidentia, which was attributed to a peculiar atrophic condition of the pelvic floor muscles, as well as those of the back and lower limbs. The infant had knock-knee and spina bifida, with the congenital deformities.

Function of the Corpus Luteum.—FRAENKEL (*Zentralblatt für Gynäkologie*, 1904, No. 19) in an interesting communication on this subject combats some of the former theories, asserting that he has frequently removed corpora lutea from both ovaries in animals, and has even cauterized the wounds, without interrupting pregnancy. He has also repeated Mandl's experiments without pregnancy resulting. He believes that corpus luteum extract has exactly the opposite action to oöphorin, and offers a better prospect of cure of osteomalacia than castration.

Ovarian Hemorrhage.—BURGER (*Zeitschrift für Geb. u. Gyn.*, Band li., Heft 2) reports the case of a nullipara, aged thirty-seven years, married for nine years, who presented all the evidences of profuse internal hemorrhage. On opening the abdomen the left ovary was found to be as large as a hen's egg, the right adnexa being normal. About two litres of fluid blood were found in the cavity. On the posterior and lower border of the ovary was a ruptured cyst, the interior of which presented a smooth surface. The ovary was generally cystic. The most careful microscopic examination revealed no evidence of ectopic gestation. A corpus luteum was present, surrounded by dilated capillaries. It was inferred that the hemorrhage was due to rupture

of some of the enlarged vessels in the wall of a corpus luteum cyst. [We recall a similar fatal case reported by Dr. Boldt, and still others in the literature. If we mistake not, excessive sexual excitement and coitus near or during the time of menstruation was held to be responsible for the accident.—H. C. C.]

Relation of Movable Kidney to Pelvic Troubles.—LEBAR (*La Gynècologie*, 1903, No. 8) found movable kidneys in 28 per cent. of 1824 patients examined with reference to this condition; 375 were right-sided, 11 on the left side, and only 4 double. The writer attributes this disparity to pressure of the liver on the right kidney, especially as the result of tight lacing, aside from the normally lower position of the right and the absence of the aponeurotic band described by Zuckerkandl on the left kidney. He believes that asthenia is the main cause of mobile kidney, hence that it has no direct relation to pelvic disorders, except as these impair the general muscular tone. [We think that it is high time that this condition is recognized as the evidence more often of general enteroptosis than a surgical condition *per se*. To treat gynecological troubles, such as prolapse of the uterus and ovaries, by performing nephropexy is generally to "put the cart before the horse." —H. C. C.]

Malignant Uterine Myoma.—STROGANOWA (*Monatsschrift für Geb. und Gyn.*, Band xviii., Heft 3 and 4) presents a paper with this title, based on twelve cases. He believes that the neoplasm is not so rare as has been supposed, having usually been mistaken for sarcoma. Rapid recurrence and metastasis prove its malignancy. It originates in the muscle cells, which degenerate and bear a close resemblance to those of sarcoma. Polymorphism and granular degeneration of the cells are the characteristic appearances under the microscope.

Such tumors are originally pure myomata, rarely fibromyomata, and should always be regarded with suspicion when they grow rapidly. [If seems as if the writer had hardly substantiated his claims that these growths are not really spindle-celled sarcomata.—H. C. C.]

Hysterectomy in Puerperal Sepsis.—ASCH (*Monatsschrift für Geb. und Gyn.*, Band xviii., Hefte 3 und 4) holds that the indications for a radical operation are exceedingly uncertain. Two points are to be considered, the character and site of the infection. The difficult point to determine is whether the streptococcic infection is localized, or has already become general. If the lymphatics are involved hysterectomy should be deferred until the result of incision and drainage of abscesses has been noted. If the veins are already thrombotic it is necessary to remove them, as well as the uterus and ovaries, the abdominal route being usually preferable. The writer reports ten cases, three of which terminated fatally, while one patient committed suicide. The other six made a good recovery.

Immediate Repair of Injuries to the Pelvic Floor.—SCHATZ (*Münch. med. Wochenschr.*, 1903, No. 44) calls attention to the fact that in forceps extractions the levator ani is apt to be torn anteriorly at its attachments to the pubic bone, and that this fact should be borne in mind in introducing the sutures—a procedure which he admits is exceedingly

difficult. It is practically impossible, when a deep tear in the posterior wall is also present, to reattach the levator to the periosteum at its original site.

The writer makes it a practice when he anticipates such an anterior tear to make a deep paraproctal incision half-way up the vagina. Even if this tear is extended it can be satisfactorily sutured.

Combined Cancer and Tuberculosis of the Ovary.—GLOCKNER (*Zentralblatt für Gynäkologie*, 1904, No. 21) reports the following unique case: A girl, aged twenty years, entered the hospital with an abdominal tumor and preliminary tuberculosis, with septic symptoms. On section a solid tumor of the ovary was removed, which presented microscopically the ordinary appearances of medullary carcinoma, but the stroma contained typical tubercular nodules and giant-cells. The opposite ovary showed many tubercles and cheesy foci. The peritoneum was absolutely free from cancerous or tubercular infiltration. The patient made a good recovery, and survived the operation nearly two years, dying of exhaustion.

The writer calls attention to the interesting fact that while three cases of primary tuberculosis of the ovary have been reported by Williams and Wolff, and that he himself has met with thirteen cases of secondary tubercular infection of ovarian cysts, there is no record of any instance in which cancer and tuberculosis were found together in an ovarian tumor.

Treatment of Hæmatocele.—ZWEIFEL (*Münchener med. Wochenschrift*, 1903, No. 34) points out the frequency of encysted hæmatoceles as compared with extensive primary intraperitoneal hemorrhages, especially from tubal ulcerations. He believes that these should be treated expectantly, though the surgeon should always be prepared for secondary rupture during the first two or three weeks. After this time the only indications for interference are unbearable pain, unusual size of the blood tumor, and infection. Vaginal section is the preferable operation, care being exercised to remove the ovum and by no means to break through into the peritoneal cavity. Under this expectant treatment patients have made an excellent recovery, and have even become pregnant subsequently.

The Relation of Fibromyoma to Cardiac Lesions.—FLECK (*Archiv für Gynäkologie*, Band lxxi., Heft 1) from a study of the material at Runge's clinic found 40.9 per cent. of cardiac diseases among 325 patients with fibroid tumors, and believes that this percentage is too small, as many affections of the cardiac muscles are hard to recognize. He infers that loss of blood is not a prominent etiological factor, since only 34.6 per cent. of cardiac cases were noted in 133 in which there was no hemorrhage. In 11 autopsies brain atrophy was noted in 8 and fatty degeneration in 3. The fact that the ovaries are so commonly affected in connection with fibroid uteri, and that excess of adipose is often observed, leads to the inference that these associated conditions may be potent, the disturbance of the "internal secretion" causing the setting free of toxins which act on the heart muscle. [While attention has been frequently called to the association of heart disease and uterine fibroid, it seems peculiar that the mortality after hystero-

myomectomy is so low, though it must be admitted that the factor of "cardiac failure" after this operation is one to be taken into account, especially in stout, flabby subjects.—H. C. C.]

OTOLOGY.

UNDER THE CHARGE OF

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The Influence of the Radical Mastoid Operation upon the Hearing.—DR. EDWARD BUHE (*Archiv für Ohrenheilkunde*, Band lvi., Hefte 3 und 4) reports from Schwartze's clinic the functional results of the radical operation in 103 cases. Of these 103 cases, 35 were improved in hearing, 37 remained the same as before the operation, and 31 were made worse. Per centum thus is: improved, 34 per cent.; remaining the same, 36 per cent.; made worse, 30 per cent. The whispered voice, that produced with the residue of air in the lungs after expiration, of a strength audible to an individual with normal hearing, 6 to 7 m., was used in testing the hearing in these cases.

From an analysis of these cases Buhe concludes:

1. The hearing after a radical operation will be found to be improved or remain the same when the labyrinth and labyrinth wall are intact, the hearing for the whispered voice before operation being less than 1 m.

2. The hearing will be improved when there is before the operation an obstruction which wholly occludes the external auditory canal.

3. The hearing will be improved or remain the same if before operation the labyrinth or labyrinth wall, or both, are diseased, there being before the operation only a small remnant of hearing.

4. There will be a decrease in the hearing when before operation the whispered voice is heard over 1 m.

5. Moreover, there will be a decrease in the hearing when the labyrinth or labyrinth wall, or both, are diseased, the hearing before operation being for whispered voice more than 0.25 m. and under 1 m.

Investigations in Regard to Caries of the Ossicles.—SCHULTZE (*Archiv für Ohrenheilkunde*, Band lx., Hefte 3 und 4) reports in detail the histological appearances of the ossicles removed from twenty-four patients. With one exception the ossicles were obtained by the radical operation. Twenty-two hammers and seventeen anvils were examined. In ten cases cholesteatomata were found in the middle ear, and in fourteen cases there were caries and granulation formation in the middle ear. The appearance of the membrana tympani before the

operation of these cases supported the truth of the diagnostic sign first brought out by Schwartze, that the site of the perforation in the upper segment of the membrana points to incus or malleus caries. The size of the perforation in the membrana gives no clue to the amount of destruction which the ossicles have suffered.

Schultze divides the pathological changes which the ossicles exhibit into two classes. In cases of the first class the diseased portion of the ossicles is on the surface and uncovered. In cases of the second class the bone change is observed beneath more or less pathologically changed tissue. In both classes the pathological process is lacunar absorption, and the covering of the carious portion with granulations, polypi, or epidermis is simply an accompaniment of the process. That the caries may come about with intact periosteum the author doubts, although the caries may start at the surface as a small point, and, as the process extends more deeply into the substance of the bone, it may become larger, and connected with the surface only by a narrow tract. The occurrence of primary osteomyelitis of the ossicles the author considers unverified. The caries is practically always secondary to a periostitis, the result of middle-ear suppuration. Loss of substance, however, may be due to pressure, as has been observed on the malleus head, where a very retracted drum membrane causes the head to press on the bony wall of the attic.

The most important immediate factor in the causation of the caries is the influence of the retained and fetid pus bathing the ossicles. This is often, although not always, associated with cholesteatoma formation in the middle ear. The immunity of the articulating surfaces of the ossicles is probably dependent upon the greater resistance of the cartilaginous coverings of these parts of the bones.

In conclusion, the author discusses the worth of ossiculectomy, and concludes that this operation is indicated in many cases, both because it often removes the focus of suppuration, and because it allows a more adequate drainage of the middle ear. The indications for ossiculectomy are, however, sharply defined, and the operation is not indicated where there is evident, in addition to the caries of the ossicles, caries of the antrum, etc., or cholesteatoma formation. Here the total exenteration of all the middle-ear cavities is indicated. The author is not in favor of leaving the ossicles intact in performing the radical operation.

Ankylosis of the Malleoincudal Joint.—Frey (*Archiv für Ohrenheilkunde*, Band lxi., S. 234–246) reviews thirty-five cases of ankylosis of the malleoincudal joint which he found in the literature on the subject. He then gave a brief clinical and histological history of two cases:

Case I. Boy, aged eleven years, had suffered with chronic otitis media for the last five years. The drumhead, with the exception of a small upper edge, was completely destroyed. The handle of the malleus lay on the inner wall of the middle ear, and the tympanum itself was filled with granulations which also surrounded the handle of the malleus. After long conservative treatment without results the radical operation was performed. At the operation the whole middle ear, as well as the mastoid, was found to be filled with granulations and pus. The bone of the mastoid was brittle. In enlarging the middle ear the malleus and incus were firmly attached and came out together.

The histological investigation of horizontal sections through the articulation showed that the bone-marrow of both ossicles was increased. The ossicles were bound to each other by a long bridge, which ran at right angles to the plane of the joint and united with the bony substance of the ossicles. This bony bridge could be followed through about half the sections. Where the bone ceased calcification began, and deeper down there was union of the ossicles, through connective tissue, which took on thickening toward the periphery.

Case II. Man, aged twenty-one years, student, suffered for several years with a frequent recurring left-sided purulent otitis media. There was complete destruction of the drumhead and of the lower part of the handle of the malleus. There were small granulations along the malleus and protruding from the attic. The outer wall of the recessus epitympanum was partly destroyed. Before a greater operative procedure an ossiculectomy was performed, at which time the hammer was removed, and with it came the incus.

The bony substance of the joint heads was greatly reduced, due to the extraordinary enlargement of the bony canals; the vessels were also enlarged and distended. The cartilage was increased. Peripherally the periosteum of the ossicles was enormously thickened.

In the first case there was union of the two joint heads through new-formed bony substance within the joint, an intra-articular ankylosis.

The enlargement of the bony canals and the vessels in them, as well as the deposit of chalk, showed the ankylosis to be the result of inflammatory processes.

The second case comes under the head of a capsular ankylosis; here, also, the result of inflammation. Frey divides cases of ankylosis of the ossicles anatomically as follows:

1. The periarticular connective-tissue ankylosis.
2. The periarticular bony ankylosis.
3. The intra-articular bony ankylosis.
4. Mixed forms in which intra-articular as well as periarticular changes exist, through the addition of connective tissue, through calcification and new-formation of bone.

Etiology. As to the cause of these changes there is too little material to determine. In twenty-three cases there existed an inflammation. This cause is held by Schwartze, Jacobson, Blau, Politzer, and others.

It is more than doubtful if catarrhal changes in the middle ear, even of long duration and combined with chronic transformation of the mucous membrane, can cause any ankylosis of the malleoincudal joint. Most cases of ankylosis occur in chronic adhesive processes after old middle-ear inflammation.

On the subject of influence of malleoincudal ankylosis upon the function of hearing not much can be said. Normally the joint makes a very small excursion, and it is even questionable if this malleoincudal articulation can be spoken of as a true joint in this respect. In these cases there are always other changes of a severe nature combined with the ankylosis, and therefore conclusions cannot be drawn as to the importance of the mobility of this articulation.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

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Hemorrhages in the Eye Present at Birth.—COBURN (*Archives of Ophthalmology*, May, 1904) has undertaken a study of this condition to ascertain if possible the cause of poor vision in certain eyes without appreciable changes in the fundus. Other observers have also reported such cases. Kenigstein, with the ophthalmoscope, found retinal hemorrhages twenty-nine times in 281 cases (about 10 per cent.). Schleich found seventy-eight eyes affected in 49 cases among 150 children, from birth to eight days old. In a week absorption had usually occurred, and only slight cloudiness of the retina remained. Bjerrum found only 3 per cent. of the cases he examined affected with retinal hemorrhages. De Vries found 11 per cent. affected in 112 cases. Of 150 newborn children examined by Montalcini from ten minutes to two days after birth, 25 per cent. had hemorrhages in the retina, 18 per cent. in both eyes.

These hemorrhages were mostly at the posterior pole of the eye. Most of these infants were in bad health. All were head presentations, and prolonged labor seemed to be a factor. These hemorrhages, occurring by diapedesis, absorb quickly, but they may often represent the origin of functional troubles.

Naumhoff examined the eyes of 47 children who had died shortly after birth, and found hemorrhages in 25 per cent. Von Hippel found hemorrhages in 10 per cent. out of 24 cases—41 per cent.

Coburn examined the eyes of 37 dead infants, either stillborn or aged up to twenty-two days. On opening the eyes the blood could often be seen with the naked eye. Hemorrhage was found in 17 cases, the retina being the chief site in all. Hemorrhage anterior to the iris is comparatively rare, having been observed but once (on the anterior surface of the iris and in the meshes of the pectinate ligament). Blood in the outer layers of the sclera was seen five times. Choroidal hemorrhage was not observed, though in one instance a detachment of the retina had evidently been caused by such. Hemorrhage into the optic nerve head was noticed four times; in the optic nerve sheath five times, and in the subhyaloid four times. The vitreous was involved only twice, though in some cases where the hemorrhage appeared to be subhyaloid only blood corpuscles were found in that structure.

A fact worthy of note is the intense congestion of the vessels and capillaries of the retina in these cases, showing that this structure is not so sparsely supplied with bloodvessels as is ordinarily supposed.

The author concludes that the probable cause of these retinal hem-

orrhages depends upon compression of the optic nerve and its central bloodvessels due to distention of the nerve sheath with cerebrospinal fluid from compression of the head during birth.

Local Anæsthesia in Eye Operations.—GUTTMAN (*Archives of Ophthalmology*, May, 1904) produces anæsthesia of the conjunctiva adequate for operative treatment of trachoma, tumors of the lid, lacrymal abscess, etc., by subconjunctival injection of a solution of cocain, 1:1000 to 1:5000 after preliminary instillation of a 4 per cent. solution or a 1 per cent. solution of holocaine in the conjunctival sac. The needle of a Pravaz syringe is introduced very superficially beneath the conjunctiva of the fornix (in the case of the upper lid the cartilage is turned with roller forceps). The entire conjunctiva is raised in a wheal; the trachoma granules thus become prominent and plainly visible, and can readily be attacked with the roller forceps without the same injury to the whole conjunctiva, as occurs in the operation as ordinarily performed.

In operations on the external surface of the lids, as for tumors, chalazia, etc., it is not sufficient to infiltrate the outer skin alone, but the conjunctiva must also be infiltrated.

The technique of the method should be thoroughly mastered.

Removal of the Crystalline Lens in Sixty-three Cases of High Myopia.—SNELL (*British Medical Journal*, February 27, 1904) reports the following results: Sixty-three operations were performed on forty-four patients, all except one under forty years of age. The degree of myopia ranged from 10 to 28 D. The operation was successful in every case. In two cases detachment of the retina occurred at intervals of six months and one year following operation. The reporter observed that of 6324 myopic eyes occurring in half the number of his private patients about 5 per cent. presented a myopia of more than 10 D. The method of operation in every case consisted of preliminary needling, followed by subsequent extraction of the disintegrated lens substance.

Increased tension, which occurs at two distinct stages, first, by reason of the swollen lens, and second, when the pupil is free or nearly so of lens matter, was successfully met by paracentesis. The writer believes that within defined limits the removal of the crystalline lens for high degrees of myopia may now be regarded as a recognized and valuable method of treatment. He does not regard detachment of the retina as discrediting the method, considering that this is a danger which always menaces eyes affected with high myopia *per se*. Patients are usually extremely grateful for the relief the operation affords.

One Hundred Cases of Eye Diseases with Bacteriological Examination.—SMITH (*Yale Medical Journal*, May, 1904) reports such a series, of which the following is a summary:

Organisms were found in all but 11 cases, of which 7 were diseases of the conjunctiva, 2 of the cornea, and 2 of other structures. There were 62 cases of disease of the conjunctiva, 15 of the cornea, 11 of the lacrymal apparatus, 9 of the lids, and 8 operative cases. The gonococcus was found in 9, and the diplococcus in 5 of the conjunctival cases without the presence of other organisms. The remaining organisms found were frequently associated with other species. The pneu-

mococcus was found in 18 cases, of which 10 were conjunctival, 3 corneal, and 5 lacrymal. Koch-Weeks bacillus was found in 4 conjunctival and 1 lacrymal case. Staphylococcus albus was present in 10 conjunctival, 4 corneal, 4 lacrymal, and 1 other case. Staphylococcus aureus was found in 2 conjunctival, 5 corneal, 1 lacrymal, and 2 others. The staphylococcus epidermidis albus was found in 1 conjunctival and 2 other cases (without other organisms). The streptococcus occurred in 2 conjunctival, 2 corneal, and 5 lacrymal, and 2 other cases. The Morax-Axenfeld diplobacillus was present in 8 conjunctival, 1 corneal, and 1 lacrymal; in the two latter with other germs. The xerosis bacillus was found twenty-three times; it occurred in a great variety of conditions. The streptobacillus was found three times in conjunctival disease. Other bacilli, diplobacilli, and diplococci were found in 28 cases. In 7 the results were unsatisfactory.

Some of these findings are suggestive. Of the staphylococcus the albus was more often found in conjunctival, the aureus in corneal disease. In the lacrymal cases the streptococcus and pneumococcus predominated.

While zinc chloride in diplobacillus, and antitoxin in Klebs-Loeffler conjunctivitis are almost specifics, none is known for any other organism. (Silver surely almost deserves the name of a specific in gonorrhœal conjunctivitis.—Ed.)

Persistent Œdema of the Eyelid Due to Syphilis.—Snydacker (*Archives of Ophthalmology*, March, 1904) reports a case of redness and œdema of the left upper eyelid occurring in a mulatto, causing the lid to hang down so as to occlude the pupil entirely seven years after syphilitic infection. There was no involvement of the tarsus and no areas of nodular infiltration in the skin or subcutaneous tissue. This condition had persisted for four months. It disappeared after three weeks of antisyphilitic treatment. Other somewhat similar cases have been described. In most of these the tarsus was involved.

The author thinks that in his case a gummatous infiltration existed in the subcutaneous tissue, or a number of small confluent or non-confluent gummatous foci were present, which because of the œdema of the lid, could not be determined by palpation or perhaps a syphilitic tarsitis or gummatous infiltration of the tarsal cartilage existed of too slight a degree to be felt.

Non-inflammatory persistent œdema of the eyelid should always awaken a suspicion of syphilis.

Diffuse Sarcomata of the Uveal Tract.—Parsons (*Archives of Ophthalmology*, March, 1904) has collected thirty-one cases of this extremely rare condition first distinguished by Fuchs in 1882. These cases show no great peculiarity in sex, eye affected, or age of patient, except the first decade, in which none of them had occurred. The most conspicuous feature of the histories is the long duration of the disease in many cases, varying, as shown by the failure of vision, from seven months to ten years. There was a history of injury of or operation in seven cases. Most of the eyes were glaucomatous. In many, indeed, the eye was excised on account of absolute glaucoma, without definite evidence of intraocular growth. Early onset of glaucoma has been emphasized, but taken in connection with the prolonged history

in many cases it is not very striking. The early onset of extraocular extension is much more striking. This is often multiple.

Pathological anatomy. These growths are all characterized by their diffuse and infiltrating tendency as opposed to the formation of a definite tumor. The cytology is particularly interesting:

1. The frequency of large round or polygonal cells, often combined with spindle cells.

2. The frequency of an alveolar or plexiform arrangement, either throughout the growths, or in parts or in secondary extensions.

3. The frequency of hyaline and myxomatous degeneration, and the occasional presence of necrosis.

4. The frequency of extension along the perivascular lymph spaces, surrounding the perforating ciliary vessels and venæ vorticosæ.

These characteristics show the cells to be endothelial and the growth endotheliomata. They vary much as regards pigmentation, which is a phenomenon of no particular value in this connection. The retina was not usually detached. Parsons thinks that the essential characteristics of these growths, their diffusely infiltrating tendency, is explained by their flatness and their endothelial origin.

DERMATOLOGY.

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Symmetrical Cutaneous Atrophy.—J. A. FORDYCE (*Journal of Cutaneous Diseases*, April, 1904) enters upon this subject very thoroughly, referring to the several well-known cases on record, and gives a case (with illustrations) of his own. The most interesting point brought out is the observation that the atrophic process, as shown by the histological examination, was plainly the result of an infiltration, and not of an idiopathic nature, the inflammatory process in the upper corium causing secondary changes in the epidermis. The alterations in the vessels' walls and the perivascular infiltration point strongly to those structures as being the primary seat of the disease. This view is strengthened by the clinical histories of such cases, the majority showing in their early stages primary vascular dilatation before the secondary atrophy was manifested.

Erythromelalgia and its Relation to Raynaud's Disease.—F. P. WEBER (*Journal of Cutaneous Diseases*, February, 1904) gives a case occurring in a woman, aged twenty-six years, free from organic disease of the central nervous system and without alcoholic or arsenical neuritis. The left foot was the part mainly affected, characterized by flushing or

lividity of the toes, and pains there and also in the left foot. Moreover, there was habitual hyperæmia of the face and tongue. The point of interest in the paper is the observation that there is no distinct line separating this disease from Raynaud's disease. The early symptoms of both forms of disease are surely of nervous origin, and can best be explained on the supposition of a vasomotor disturbance due to the central nervous system. In both this disease and in chronic Raynaud's disease the muscular coats of the arteries become hypertrophied. Clinically, intermediate cases between the two diseases are often met with in literature, as Cassiner has shown in his work on vasomotor trophic neuroses, published in Berlin in 1901.

Lepra in the Sandwich Islands.—DALAND (*Journal of the American Medical Association*, November 7, 1903) is of the opinion, from his observations, that the Hawaiian is unusually susceptible to this disease. He also regards syphilis as a predisposing cause. While heredity is a factor, it is of minor importance. The disease he considers but feebly contagious and to be incurable.

On the other hand, Filaretopoulo, of Athens (*British Journal of Dermatology*, September, 1903), holds the belief that the disease is hereditary and seldom contagious, and that the treatment with mercurial injections and chaulmoogra oil, up to 300 drops daily, together with hygienic and dietetic regulations, are useful.

A Clinical and Histological Study of Folliclis.—ALEXANDER (*Archiv für Dermatologie und Syphilis*, Band lxx., Heft 1) concludes from a study of the histories of seven cases of folliclis that there is an undisputable relationship between this affection and tuberculosis. The nodules occur in crops, and are apt to recur at certain seasons of the year, sometimes returning every spring, or occurring only in the summer or winter. It is noteworthy that a considerable proportion of patients complain of joint-pains for which there is no clinically demonstrable cause. In some cases œdematous, painful infiltrations occur, which are due to the coalescence of several deeply situated nodules. While folliclis is an exquisitely chronic affection, there are considerable periods during which the patient shows nothing but scars. It is also distinguished by the multiformity of the lesions, which may at times be so insignificant as to escape the patient's attention and to be readily overlooked by the physician. A knowledge of this apparently in itself insignificant affection may be of great importance, since by its recognition we may be able to destroy a visible focus of infection which is a constant source of danger to the entire organism.

Histologically, the author distinguishes two types of the disease: a deep variety with tubercular change, and a superficial form with diffuse inflammatory infiltration—thrombophlebitis. Both arise, without doubt, through embolic processes, and certainly are due to tubercle bacillus itself.

The Relationship of Alopecia Areata to Dental Irritation.—BETTMANN (*Archiv für Dermatologie und Syphilis*, Band lxx., Heft 1) has studied thirty-three cases of alopecia areata, observed in the Heidelberg medical clinic, for the purpose of determining the correctness of Jacquet's theory concerning the dental origin of this affection. As the

result of the careful study of these cases he believes it would be a mistake to accept Jacquet's hypothesis unreservedly. In none of these thirty-three cases was it possible to demonstrate that the alopecia had been preceded by dental irritation.

Herpes Zoster Associated with a Generalized Bullous Eruption from Prolonged Administration of Arsenic.—SEQUEIRA (*British Journal of Children's Diseases*, vol. i., No. 4) reports the case of a girl, aged five years, to whom three minims of Fowler's solution were given three times a day for an attack of guttate psoriasis. After three months of this treatment pigment began to appear upon the trunk, and a month later the skin showed characteristic arsenical pigmentation. An eruption of vesicles and bullæ now appeared all over the trunk, the face and extremities being exempt, and at the same time an abundant zoster appeared in the lumbar region, on the right side. No other arsenical symptoms, such as palmar and plantar hyperkeratosis, muscular disturbances, or gastric catarrh, were noted. The herpes was followed by scarring, but the vesicles and bullæ upon the trunk left no scars. The author regarded the eruptions as arsenical in origin.

Eosin in the Treatment of Lupus.—LEDERMANN (*Archiv für Dermatologie und Syphilis*, Band lxx., Heft 1) at a meeting of the Berlin Dermatological Society held February 2, 1904, presented a patient with lupus who had been treated by applications of eosin according to the suggestion of Jesionek and Tapeiner. The applications had been made several times a day, and it was clearly apparent that the eosin had an elective action upon the lupus nodules, since not only the ulcerated surfaces but the superficial nodules showed evidences of destruction. In a case of cancroid of the skin there was also tissue destruction. It can be said that dyeing with eosin produces an effect similar to pyrogallol upon lupus nodules, but does not cause pain.

A Case of Paget's Disease of the Umbilicus.—T. COLCOTT and J. M. H. MACLEOD (*British Journal of Dermatology*, February, 1904) report a case of Paget's disease occurring in a most unusual region. The patient was a man, aged sixty-five years, who presented a rounded, eczematoid patch about two inches in diameter, occupying the umbilical region. This patch, which had been forming for about eleven years, was a brilliant red color, ulcerated, covered in spots by epithelium, and surrounded by a raised, well-marked, sharply defined border. The clinical diagnosis was confirmed by the histological examination. The changes found in the epidermis were typical of the disease, and the authors believe that the histological alterations in the epidermis in Paget's disease are characteristic and pathognomonic, whatever the region affected. No evidence of definite malignant change was found in the epidermis. The authors are not inclined to accept the view that the epidermic changes are malignant from the beginning. The histological appearances were suggestive of a chronic inflammatory process affecting the papillæ, leading to proliferation, œdema, and degeneration of the cells of the epidermis, in some respects resembling lupus erythematosus. The authors' researches shed no light upon the cause of the disease.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Physiology of the Thyroid Gland.—KISHI (*Virchow's Arch.*, 1904, Band clxxvi. p. 260) describes a series of observations dealing with the extirpation of the thyroid gland in animals and upon the metabolism and the pathological changes in the various organs of thyroidectomized animals. From a study of the parathyroid glands after extirpation of the thyroid itself, he believes that the parathyroid is not an independent organ, but an embryonal thyroid rest, and under certain circumstances may be transformed into thyroid tissue. Dogs and cats often die after thyroid extirpation, even though the parathyroids are left intact. If the thyroid gland is normal the parathyroids are functionless, and they exert a function only when the thyroid is entirely removed or when that portion of the gland which remains is not sufficient to sustain life. When the quantity of parathyroid is equal or exceeds the quantity of thyroid necessary to sustain life a thyroidectomized animal does not die.

It was further found that in thyroidectomized animals metabolism was increased, while there was no change in control animals in which the thyroid gland had simply been exposed at operation. The amounts of nitrogen, sulphuric acid, phosphoric acid, and uric acid eliminated in the urine followed the same curves. The chloride excretion was very irregular.

The changes in the organs of the thyroidectomized animals were confined chiefly to the blood and bloodvessels. A direct result of thyroid extirpation was a decrease in hæmoglobin and red blood cells. The alterations of the other organs described by many authors was thought to be of secondary nature.

The author concludes from these studies that the thyroid is a secretory organ, and neutralizes within the gland an albumin that is injurious to the blood. This toxic substance is a variety of nucleoproteid derived from the cell nuclei of food. In the glandular cells of the thyroid an iodine containing globulin is formed, which possesses an attraction for the albumin of the toxic nucleoproteid. These two substances in combination are eliminated from the cells into the follicles as a third substance which the author calls "thyreotoxin." Thyreotoxin is split into two harmless products which gain access to the lymphatics and bloodvessels, and are thus eliminated.

Researches upon Chronic Passive Congestion of the Liver.—HART (*Ziegler's Beiträge*, 1904, Bd. xxxv. p. 303) has examined the liver in 35 cases of advanced central red atrophy. He finds constantly in this

condition thrombosis of the capillaries toward the central portion of the liver lobule. The capillary plugs are to be seen as soon as the degeneration of the liver cells makes its appearance, and the extent of the thrombosis is in direct proportion to the degeneration of the liver cells. The production of the thrombi forms a new argument in favor of Schmidt's theory concerning the relationship between fibrin coagulation and cell degeneration.

Upon the Properties and Production of Anticrotalus Venin.—FLEXNER and NAGUCHI (*Journal of Medical Research*, 1904, vol. xi. p. 363) have shown in previous studies that the severe local lesions (mainly hemorrhage and softening of the tissue) which follow injections of rattlesnake venom are produced in large part by a cytotoxin which destroys the endothelial lining of the bloodvessels. To this toxin they have given the name of hemorrhagin. Owing to the difficulty in overcoming these local effects of the venom no successful production of antitoxin for rattlesnake venom has so far been accomplished. The present work deals with a method of surmounting this difficulty. By treating the crotalus venom with dilute hydrochloric acid or iodine trichloride, the locally active principles could be transformed into toxoid substances. This modified venom, though deprived of a large part of its toxicity, still preserved its power to set up antivenin formation in the rabbit and dog. Thus, by injections of this modified venom an antivenin of considerable activity against rattlesnake venom could be procured. While the toxophore group of hemorrhagin was primarily attacked by the weak hydrochloric acid and iodine trichloride, both the toxophore and cytophilic haptophore groups were destroyed by strong hydrochloric acid and hydrochloric acid and pepsin, thus rendering the substance useless for antivenin production. Iodine trichloride destroyed both the groups in the neurotoxins and hæmolysins of cobra and daboia venoms.

Anticrotalus venin was found to be without appreciable antitoxic power over cobra and daboia venoms, and of imperfect antitoxic power over water moccason venom. This was in keeping with the different constituents of these venoms, for the first two owe their toxicity to neurotoxins and hæmolysins, the last to these principles together with hemorrhagin.

Precipitins were formed from venom along with or independent of the immunizing principles for venom, but there was no relation between the degree of protection afforded and the amount of precipitins present in the immune serum. The precipitins for the different venoms were highly though not absolutely specific.

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